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AFEM HANDBOOK OF ACUTE AND EMERGENCY CARE

- The only handbook for emergency care in sub-Saharan Africa
- How to deal with conditions in settings with limited or with full resources
- Endorsed by the African Federation for Emergency Medicine

AFEM
African Federation for Emergency Medicine
Fédération Africaine de Médecine d'Urgence

Lee A. Wallis
Teri A. Reynolds

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List of abbreviations

🕮	cross reference
◇	moderately resourced setting
◈	fully resourced setting
↑	increased
↓	decreased
½ DD	½ Darrows Dextrose 5%
adrenaline	epinephrine
ALoC	altered level of consciousness
ABG	arterial blood gas
AMS	altered mental status
AS	aortic stenosis
AXR	abdominal X-ray
BB	beta-blocker
BID	twice daily/Q12h
BVM	bag valve mask
Ca	calcium
cardiac monitor	ECG monitor
CBC	complete blood count/FBP/FBC
CCB	calcium-channel blocker
CCF	congestive cardiac failure
chest tube	intercostal chest drain
CMV	cytomegalovirus
CNS	central nervous system
crepitations	crackles/rales
CTG	cardiotocogram
CXR	chest X-ray
D5%	5% dextrose
DDX	differential diagnosis
DIB	difficulty in breathing, SOB
DM	diabetes mellitus
EBV	Epstein Barr virus
GA	general anaesthesia
GBS	Guillain Barré sydrome
GCS	Glasgow Coma Score
GSW	gunshot wound
Hgb	haemoglobin
HIV	Human immunodeficiency virus
HSM	hepatosplenomegaly
HSP	Henoch-Schönlein purpura
IVF	intravenous fluids
JVP	jugular venous pressure
K	potassium

labs	laboratory investigations
LAD	left axis deviation
LAN	lymphadenopathy
large bore IV	approximately 14G–16G intravenous access
LBBB	left bundle branch bock
LFT	liver function tests
LMWH	low molecular weight heparin
LoC	loss of consciousness
LA	local anaesthetic: lidocaine/lignocaine
LV	left ventricle
LVH	left ventricular hypertrophy
MR	mitral regurgitation
MS	mitral stenosis
MVC	motor vehicle collision
Na	sodium
NIPPV	non-invasive positive pressure ventilation
NPO	nil by mouth (nil per os)
O_2	oxygen
O_2 sat	oxygen saturation
PE	pulmonary embolism
POC	point of cure
PPV	positive pressure ventilation
PR	per rectum
PRN	pro re nata ‘as the circumstance arises’
PT/PTT	coagulation
PTX	pneumothorax
PUD	peptic ulcer disease
Q4h	every four hours
Q6h	four times daily/QID
QD	daily, once daily
RAD	right axis deviation
RBBB	right bundle branch block
ROM	range of motion
RV	right ventricle
SIRS	systemic inflammatory response syndrome
SLE	systemic lupus erythematosus
SSSS	staphylococcal scalded skin syndrome
SXR	skull X-ray
TB	tuberculosis
TID	three times daily/Q8h
TR	tricuspid regurgitation
type and cross	ABO and Rh compatibility testing
UA	urine analysis
URTI	upper respiratory tract infection

US	ultrasound
VBG	venous blood gas
VS	vital signs
WBC	white blood cells
XR	X-ray

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We are very grateful to RTT Logistics for their invaluable support in helping us to distribute 8 400 copies of this handbook across 44 countries.

Dedication

LW – for Abbi, Francis and Ava, who make it all worthwhile.

TR – for Kai (even though he prefers big books); and for Franco, *fabbricatore della felicità*.

Part

1

Introduction

Introduction

'Emergencies occur everywhere, and each day they consume resources regardless of whether there are systems capable of achieving good outcomes.' Bulletin of the WHO 2005; 83(8):626–631.

Every day across Africa there are millions of medical emergencies. You are probably reading this handbook because you care for the people who are affected. You may be a nurse or a doctor, a medic or a clinical officer; you may see such patients every day, or only rarely; you may work on an ambulance, in a clinic, or in any level of hospital, and you may or may not have an area dedicated to the care of emergency patients (casualty, emergency centre, emergency department, or trauma unit). Whoever you are and wherever you work, every day people need your help with their emergencies.

This book is here to help you do your job. It is easily accessible and practically orientated. It includes Rapid Assessment Protocols (RAPs) formatted on two facing pages, designed to lay open and guide care at the bedside if needed, as well as more detailed chapters on specific conditions.

We acknowledge that there are many ways to approach clinical problems, and that there is often no definitive answer as to which way is best. In this handbook, we have aimed to provide an approach that works, and that is context-appropriate. You can adapt our recommendations into your clinical context to find the best solutions for your patients.

An emergency care handbook for Africa

The book has particular features for the emergency care provider working in Africa. It contains specific detail on local diseases, for example; it doesn't assume specialist physician availability; and it provides a presentation-based approach to guide care even when definitive testing is not available.

We acknowledge the reality that emergency care is being delivered every day in a wide variety of settings with a range of available resources. Where possible, we have tried to identify appropriate investigations and effective treatments that are available at each resource level, rather than limiting our recommendations to interventions available only in a small number of tertiary sites. While this can never be exact, and actual resource availability will vary from site to site, we have designated the level of resources usually available in a well-stocked district or small regional hospital as Moderate (indicated by ◇), that of a central (national) hospital or large regional or as Full (indicated by ◈). In general, unmarked investigations or interventions are widely available at all resource levels.

Should you wish to use this book as part of a broader emergency care educational programme, it is coordinated with the open-access AFEM emergency care curriculum and associated presentation bank (available at www.afem.info).

Book division

This book is divided into eight sections. Throughout you will see cross references to other relevant chapters (indicated by 🕮). Our paediatric recommendations are orientated towards those aged 12 years and younger.

Section 1 explains how to use this book and describes some general principles of emergency care. Section 2 provides an algorithmic approach to guide the first few minutes of managing the undifferentiated critically ill adult,

child or neonate. The Section 3 RAPs build on this management in a syndrome-based way, leading you quickly through the initial clinical approach (including resuscitation and stabilisation) and directing you to relevant chapters in the rest of the book. These protocols are provided as two page algorithms. See 🕮 p. 4 for more information on the RAPs.

Section 4 addresses specific conditions by body system. Inevitably, many conditions may affect more than one system, and there are certainly other possible classifications we could have used, but we hope that this structure, in conjunction with the detailed index, will allow you to find what you are looking for quickly and easily.

Section 5 provides a rapid review of basic bedside ultrasound applications in the acute care setting. This section is not intended to be comprehensive, or to replace practical training, but to rapidly remind trained providers of the key points for each application. Section 6 covers the most common practical procedures undertaken in emergency care. Each chapter guides you through the relevant indications and contraindications, preparation (including the key risks and benefits for each procedure to facilitate informed consent), actual procedure steps, and necessary aftercare.

Section 7 addresses structural aspects of the emergency care system, including out-of-hospital care, disaster response, communication, and the approach to special patient populations. Section 8 includes useful reference materials and commonly used abbreviations, in addition to a chapter on useful sources of additional information to supplement the material in this handbook.

We hope that there will be many future editions of this handbook, and would value any suggestions you have to help improve it. Please direct any feedback to programs@afem.info.

Philosophy of emergency care

Hippocrates famously said, 'Life is short, art is long,' but less famously, he continued, '…the crisis fleeting, experience perilous, and decisions difficult'. Peril, crisis and difficult decisions are the mainstay of emergency care, which we define as:

> The provision of initial resuscitation, stabilisation, and treatment to acutely ill and injured patients, and delivery of those patients to the best available definitive care.

The goal of emergency care is to get people safely from the world into the hospital, from the site of acute injury or illness to definitive care. There are emergency care components at every level of the health system, from bystander response through to tertiary interventions. Emergency care is a system that covers areas from the community and the field, to the OT and the ICU.

Emergency care is inherently political, as the practice itself forges system links, but also because the political work of juggling human and other resources to manage patients is a daily essential.

Emergency practice exists within a network of seeming contradictions. The breadth of the practice necessitates both humility (you must know what you do not know), and hubris (your scope is the initial management of everything). It is a practice of extreme individual accountability, inevitably embedded within a team. The field is characterised by rapid diagnosis, but also by the need to treat patients who never get a diagnosis. Emergency providers learn to treat constellations of signs and symptoms. Emergency interventions demand precision, as well as a tolerance for approximation that allows immediate action. Anticipation and preparation are as valuable as the willingness to improvise in the face of the unexpected.

Appropriate actions for a given situation are defined as much by the patient in front of you, as by the conditions around you. Care is always delivered to the individual, but often with an eye to optimising outcomes for the group (the department, a crowd, mass casualties etc.). Patients are not usually treated in the order in which they arrive, but in order of acuity. Acuity is not the same as severity of illness, but results from a combination of the illness itself and the potential for intervention that will change its course. Triage is this process of prioritising treatment that will make a difference and is essential to the philosophy of emergency care.

Like other health care practitioners, emergency care providers approach patients by gathering information from the patient history and physical examination, and using that information to guide tests and interventions. The specificity of emergency practice is the need for this process to be iterative, the need to repeat the cycle multiple times,

increasing the depth and level of detail as a patient is stabilised. While we often discuss cases as a linear progression from a single history and physical to comprehensive diagnostics and therapeutics, actual emergency practice is different: an immediate brief history and physical are followed by critical diagnostic tests (e.g. vital signs, oxygen saturation and rapid blood glucose) and immediate treatments (e.g. airway repositioning or IV fluids). This first cycle of history, physical examination, diagnostics and therapeutics constitutes the ABCs or primary survey. Once this primary survey has been completed, another cycle begins: a more in-depth but still focused history is sought, and is followed by a complaint-orientated physical examination, further rapid testing (point of care ABG or ultrasound), and further time-critical interventions (chest tube, or anti-seizure medications). Ultimately, in later cycles, a complete history and physical examination are done and laboratory and imaging investigations are completed. The first cycle is usually a matter of seconds to a few minutes, the second occurs over many minutes, and the rest may take hours.

How to use the Rapid Assessment Protocols (RAPs)

The Rapid Assessment Protocols in this volume aim to capture this reality of emergency care practice and provide a map to lead providers through the elements of the history, physical examination, diagnostics, and therapeutics that are essential immediately, within the first 5–15 minutes, and within the first hour. The RAPs are printed across two facing pages designed to be laid open at the bedside to lead providers rapidly through early management and help direct to appropriate chapters within the book.

Ultimately, emergency care providers need a high tolerance for acuity, uncertainty and contingency. They are rewarded with the daily opportunity to accompany patients through extraordinary events, to work in a team that transcends the capabilities of any of its members, and to change the course of illness.

Part
2

Approach to the unstable patient

1 Adult resuscitation: Basic concepts and practice

Any approach to resuscitation needs to be systematic, simple and robust to allow the resuscitator to act even with limited information.

The ABCs of resuscitation

The ABCs provide a framework for the evaluation and treatment of severely ill patients:
A – Airway: check for any obstruction to passage of air to the lungs
B – Breathing: ensure adequate movement of air to the lungs (via negative or positive pressure)
C – Circulation: evaluate whether cardiac output provides adequate perfusion to deliver O_2 to the tissues
D – Disability: assess and protect brain and spine function
E – Exposure/environment: identify all injuries and any environmental threats to patients and staff (hypothermia, chemical exposures etc.)

Initial resuscitation involves rapid assessment and early intervention in each of these essential systems. A lone rescuer would do this in series; more than one rescuer could intervene in parallel.

Although we list the items alphabetically here, current resuscitation guidelines follow the sequence C-A-B, as cardiac causes for arrest in adults are much more common than primary airway problems. Research indicates that good quality chest compressions and early access to defibrillation are the most important initial factors influencing resuscitation outcomes. There is less consensus in children with some authorities advocating ABC and others CAB: check your local protocol.

A – Airway

Assessment

Assess for evidence of airway obstruction:
• Look in oropharynx for swelling, secretions or other objects causing obstruction
• Noisy or abnormal breathing sounds
• Inability to speak or change in voice
• External evidence of trauma or compressive masses

Demonstration videos can be found on the YouTube channel EM Cape Town, or at:
http://www.youtube.com/channel/UCDP5VfSnQ3AB1xgPbMUTniw

Intervention

Position and clear the airway:

- If injury of the cervical spine is suspected, prevent movement of the neck using manual immobilisation or rigid cervical collar and or head blocks
- Open the airway using the jaw thrust or head-tilt-chin-lift manoeuvre
- Remove liquids (blood, secretions) using suction with a rigid catheter
- Remove foreign bodies under direct vision with forceps (i.e. McGill's forceps).Avoid blind finger sweeps

Now re-assess the airway and attempt a rescue breath if apnoeic. If still obstructed:

Basic equipment and devices:

- Nasopharyngeal airway
- Oropharyngeal airway

Place the device and re-attempt rescue breaths. If still obstructed:

Advanced equipment and devices:

- Non-definitive airway devices:
  - » LMA, laryngeal tube
- Definitive airway devices:
  - » ET tube
- Difficult airway assist devices and techniques:
  - » External airway manipulation
  - » Gum elastic bougie, intubating stylet, intubating LMA
- Surgical airway
  - » Open cricothyroidotomy (ad-hoc or commercial sets), retrograde intubation
  - » Needle cricothyroidotomy is unlikely to provide much benefit

Management of choking

Choking indicates airway obstruction by a foreign body, usually above the vocal cords. If the obstruction is complete, choking is rapidly fatal. The most common cause is food, with small objects and toys an important cause in children.

If someone appears to be choking, and the patient is **awake/responsive,** follow these steps:

Step 1:

- Call for help.

Step 2:

- If they can still move air (partial obstruction) encourage them to cough and try to expel the object.

If they cannot…

Step 3:

- Perform the Heimlich manoeuvre to assist with expulsion of the object. Stand or sit behind the patient. Wrap your arms around his/her body. Place your dominant hand as a fist in their upper abdomen (just below the xiphisternum) and wrap your fist with your other hand. Now pull your hands toward your own chest in fast, firm movements. These inward jerks will hopefully force air out of the lungs with enough force to expel the object.
- Continue until the object is out or the **patient becomes unresponsive ...**

Step 4:

- Lay the person down on a firm surface.

Step 5:

- Initiate CPR.
- Start by opening the airway and looking for an easy to reach foreign body. If visible and reachable, remove it and give two rescue breaths.

- If no FB is visible, attempt two rescue breaths and then start chest compressions. Do not attempt blind finger sweeps. If you have a laryngoscope and long forceps, consider removal of the object under direct vision.
- Now continue standard CPR, checking for a FB after every 30 compressions before delivering breaths. Even if you cannot see a FB, attempt to deliver breaths as the CPR may have dislodged the FB.

Step 6: (may consider at any stage if the patient becomes unresponsive)

- If you have the training and equipment, perform a surgical airway procedure and establish a definitive airway if the FB cannot be dislodged.

Rapid sequence intubation (RSI)

Intubation is dangerous and should only be undertaken if the benefit to the patient outweighs the risk of harm. RSI is a sequence of steps used to secure the patient's airway by intubation with a definitive airway device – the endotracheal tube (ETT). RSI establishes a definitive airway, but you must continue resuscitation, investigation and treatment.

Prepare the patient

If possible, explain to the patient what is about to happen. Ensure good IV access. Position the patient somewhere you can manipulate the head, neck and torso.

If time allows, assess the likelihood of a difficult airway:

L – Look for external masses, obesity, receding mandible, injuries etc.
E – Evaluate via 3-3-2 rule:

- Adequate mouth opening: 3 of the patient's fingers
- Hyoid-chin distance: 3 of the patient's fingers
- Thyroid-floor of mouth distance: 2 of the patient's fingers

M – Mallampati score (I, II – uncomplicated; III, IV – difficult)
O – Obstruction (swelling, foreign bodies, haematomas, tumours etc.)
N – Neck mobility

Provide high flow O_2.

Prepare the equipment

Equipment will be dependent on context and resources, and should be gathered to the bedside and checked.

Minimum equipment:

- Suction source, tubing and catheters
- O_2 source and tubing
- BVM with a selection of masks
- OPA, NPA
- An induction agent and paralysing agent
- A working laryngoscope (check bulb, batteries, and handle opening)
- A selection of endotracheal tubes with a syringe to inflate the cuff (check cuff for leak)
- Some form of assist device: introducer/stylet, bougie etc.
- Tape to secure the ETT
- Equipment to perform a surgical airway if needed
- Monitoring equipment – SaO_2 and cardiac monitor

Further equipment (if available):

- Alternative airway devices such as LMA, pharyngeal tube, light wand etc.
- Capnometer
- Naso-gastric tube and bite block
- Non-invasive ventilation or CPAP capabilities
- Video laryngoscope etc.

Note that if you are practising in austere environments, you may have to make do with sub-optimal equipment, which may change the balance of potential risk and benefit to the patient. Whether or not intubation is indicated will depend on available equipment and on available resources for post-intubation care. Understand the procedure and its risks well so you can improvise intelligently.

Pre-oxygenate

Aim to maximise Hb saturation, PaO_2 and O_2 concentration before attempting intubation. This O_2 'buffer' allows for a longer apnoea time before the patient becomes hypoxic.

- If the patient is breathing spontaneously: 3 minutes of 100% O_2 by high flow face mask
- If you are assisting breathing: 8 vital capacity breaths with 100% O_2 and an assist device (BVM)

When available, also use continuous nasal prong oxygenation throughout the procedure, even when ventilation is paused during intubation.

Pre-treatment medications

Pre-treatment is controversial and many practitioners leave it out completely. Pre-medications may be useful in certain cases if administered at least **three** minutes before intubation, **but** pre-medication should never delay a definitive airway in a critical situation. If delay will not endanger the patient, consider:

- Fentanyl (1–3 mcg/kg IV)
  - » If raised intracranial pressure, ischaemic heart disease, aortic dissection or AAA
- Lignocaine (1.5 mg/kg IV)
  - » For suspected raised intracranial pressure or in severe bronchospasm (unclear evidence for benefit)

Induction

The ideal induction agent would rapidly induce anaesthesia and maintain airway-protective reflexes while not causing haemodynamic problems. Use what you have and are familiar with. Etomidate and ketamine are effective agents with excellent safety profiles.

- Etomidate: 0.3 mg/kg IV:
  - » Unlikely to cause hypotension
  - » Theoretical concerns regarding use in sepsis not supported by research
- Ketamine: 2 mg/kg IV:
  - » Unlikely to cause hypotension. Preserves airway reflexes. Wide therapeutic index
  - » Previously thought to be harmful in head injury; may in fact be the agent of choice
  - » Safe to use in children
- Thiopentone: 3–5 mg/kg IV
- Midazolam: up to 0.3 mg/kg IV

Use extreme caution with thiopentone or midazolam (especially in patients who are hypotensive, hypovolaemic or have head injury) due to pronounced hypotension and respiratory depression at induction doses.

Paralysis

Administer the paralysing agent immediately after the induction agent.

- Succinylcholine 2 mg/kg IV (consider 1.5 mg/kg in adults):
  - » Depolarising muscle relaxant: expect muscle fasciculation
- Rocuronium 1–1.2 mg/kg IV:
  - » Non-depolarising agent: no fasciculation

Once the induction and paralysing agents have been given, stop ventilation, position the patient, consider cricoid pressure and await paralysis. The patient is ready for intubation once the mandible can be opened and closed freely and without muscular resistance (the 'slack jaw' test).

Intubation

Intubate the patient using a laryngoscope and the correct size ETT. If initial attempts fail, ventilate the patient for 5–8 breaths to restore oxygenation and attempt again, considering assist devices and techniques (external airway manipulation, bougie, introducer etc.).

Primary confirmation of position should at least include 5-point auscultation (both axillae, both anterior lung fields, epigastrium). Detection of end-tidal CO_2 is highly desirable if available.

Post intubation management

Secure the ETT with ties, tape or a commercial device. Initiate manual or mechanical ventilation. Obtain a chest XR. Consider placement of a nasogastric tube and bite block. Consider sedation using bolus or continuous infusion techniques.

Continue resuscitation, treatment and investigation of the patient. Consult the in-hospital ICU and definitive care teams.

B – Breathing

Once the airway is secure, assess and manage breathing.

Assessment

Assess for the presence of breathing (agonal respiration should be managed as apnoea). If present, assess for adequacy (if the patient is hypoxic or displaying evidence of respiratory distress, breathing is not adequate).

In breathing patients, a brief initial evaluation should look for: tracheal deviation, asymmetrical percussion note, decreased or abnormal breath sounds, and evidence of trauma.

Intervention

Supplemental O_2:

- Provide supplemental O_2 to all critically ill patients initially, particularly those who have A or B problems. Maintain oxygenation in the normal range for the patient: cyanosis should resolve and, if available, SaO_2 measurement should show improving oxygenation (> 92%)
- Methods include: nasal prongs, simple face masks, adjustable venturi face masks, partial rebreather face masks and non-rebreather face masks

Rescue breaths:

- If no equipment is at hand, a rescuer can provide rescue breaths with mouth-to mouth breathing, ideally using a barrier device (face shield, pocket mask)

Basic equipment and devices:

Remember to use supplemental O_2 if available.

- Face mask
- Self-inflating bag-valve-mask ventilation device (BVM)

If ventilation is difficult, consider airway problems and re-assess the airway.

Advanced equipment and devices:

- Non-invasive ventilation devices:
  - » Should not be used for patients with cardiac arrest
  - » Useful in specific cases of respiratory distress/failure
- Mechanical ventilation:
  - » Not useful during chest compressions

Mechanical ventilation

A patient with absent on inadequate respiration requires ventilation assistance. BVM ventilation is the simplest technique. Once the patient is intubated and requires ventilation for a prolonged period, manual ventilation becomes

impractical. Such patients should be connected to a mechanical positive pressure ventilator. Note that during CPR and chest compressions a mechanical ventilator will not work – continue with the BVM until ROSC is achieved.

There are many different machines, modes and techniques for mechanical ventilation and a full discussion falls outside the scope of this chapter. It is critically important that you get to know the machine available to you.

For details of setting up ventilators, see p. 811.

Patients who are being ventilated require analgesia AND sedation by either an infusion or bolus regime. Follow your local protocol.

All patients on positive pressure ventilation (including bag-valve mask) are at risk of pneumothorax. Serial clinical and possibly XR or PTX evaluation is needed to monitor for PTX. Always consider PTX in the worsening patient – positive pressure ventilation can covert a small or previously occult PTX to a tension PTX.

C – Circulation

The most common cause for adult non-traumatic arrest is primary cardiac arrest, usually due to ischaemia. Rapid assessment and management of C problems is the most important aspect of CPR.

Assessment

Feel for a (central) pulse for no more than 10 seconds. If absent, start CPR at once with chest compressions and use the AED/defibrillator as soon as possible.

If the pulse is present, look for signs of circulatory compromise:

- A pulse that is weak and either too fast or too slow
- Hypotension
- Evidence of poor peripheral perfusion: cold and clammy peripheries, prolonged capillary refill time
- Evidence of poor organ perfusion: AMS, tachypnoea, decreased urine output

Intervention

Management of C problems depends on whether cardiac arrest is present or not. Patients in cardiac arrest need CPR and rapid access to an AED or defibrillator. Patients with shock need rapid assessment and correction of the underlying cause.

The tools of 'Circulation' management include: chest compressions, AED or defibrillator, IV access for fluids and drugs, devices and drugs aimed at the underlying cause.

If the **pulse is absent**, start CPR at once and get an AED/defibrillator to assess the arrest rhythm (see Ischaemic heart disease, (p. 110), Cardiogenic shock, (p. 88):

- If 'shockable' (VF, pulseless VT, torsades du pointes): defibrillate and resume CPR (p. 930)
- If 'non-shockable' (asystole, PEA): continue excellent CPR and find and reverse the cause

If the pulse is present, look for signs of shock and circulatory compromise (as above).

Further management should proceed according a resuscitation algorithm (Figure 1.1 on the next page) until return of spontaneous circulation and then focus on finding and treating the cause of the arrest.

D – Disability

Disability refers to a rapid assessment of potential neurological problems. Note that many CNS problems cannot be 'fixed' during resuscitation, but it is essential that they are identified prior to paralysis for intubation, as some spinal and brain injuries have substantial implications for the management of basic haemodynamics. Excellent CPR, prevention of secondary brain injury and early involvement of the definitive care team is essential, and there are a few interventions during the resuscitation phase that significantly increase the chance of favourable neurologic outcome.

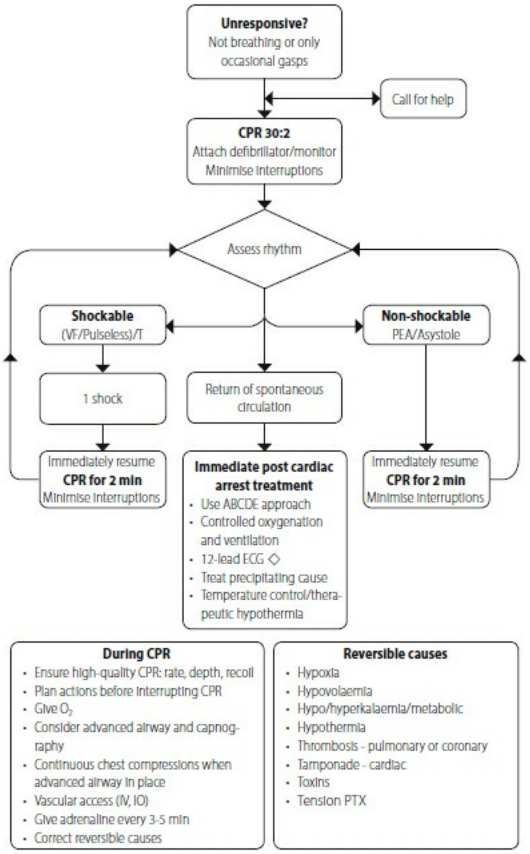


Figure 1.1 Resuscitation algorithm

Assessment

Assess the level of consciousness using AVPU (or GCS in trauma patients). Evaluate for focal neurological deficit – pay particular attention to the pupils. Note any seizures. If possible, look for raised intracranial pressure.

Intervention

Consider:

- Specific measures:
  - » Correct hypoglycaemia
  - » Terminate seizures
- Prevent secondary brain injury (specific measures plus:):
  - » Effective CPR with monitoring of pulses during compressions
  - » Ensure adequate ventilation and oxygenation
  - » Ensure adequate brain perfusion
  - » Head up 30° if possible
  - » Protect the airway if necessary
  - » Early recognition of primary CNS pathology and involvement of the definitive care team

The most important part of D management is A, B, C management.

E – Exposure

Assess the patient for hypothermia or hyperthermia. Completely undress and carefully examine to find evidence of injuries, toxic exposures, exposures to allergens (such as bee stings), needle marks etc. Prevent hypothermia by covering the patient as soon as possible.

Conclusion

Resuscitation is a core skill for any health care provider, particularly those involved in emergency care. We often need to act rapidly with limited information, time and equipment. Learning, practising and using a structured approach to resuscitation and CPR is an essential part of emergency care.

Remember that good initial management and stabilisation of very ill patients may make full resuscitation and CPR unnecessary. Avoiding the need for ABC resuscitation is the primary goal. Also remember that return of spontaneous circulation is not the end goal but only creates the possibility of return to function.

2 Paediatric resuscitation: basic concepts and practice

The basic principles of resuscitation of children beyond the neonatal period are similar to those for adults, though with some important differences. This chapter will highlight these and present an algorithm for paediatric resuscitation. It is meant to be used in conjunction with Adult resuscitation: Basic concepts and practice (p. 8).

Children tend to be more resilient initially in the face of physiological stress than adults, but once decompensation begins, it is rapid and severe.

Assessing the critically ill child

See also RAP section for assessment of specific complaints (pp. 29–81).

- **General assessment**: rapid (over seconds) observation of appearance, work of breathing and adequacy of circulation using visual and auditory cues
- **Primary assessment**: a rapid ABC assessment, including VS and monitors (if available)
- **Secondary assessment**: a focused history and clinical examination performed after ABC stabilisation
- **Tertiary assessment**: full examination and further advanced diagnostics as needed

General assessment

A rapid assessment by visual observation and listening that should take only seconds and identify unstable patients in need of immediate intervention. This assessment takes place as you walk to the bedside.

- Observe muscle tone, level of interaction, consolability, speech/cry and colour
- Observe the work of breathing: note respiratory distress (tachypnoea, recessions/retractions, grunting, alar flare, cyanosis) and evidence of possible airway compromise (noisy breathing, audible wheeze, stridor)
- Assess circulation: skin colour and temperature, haemmorrhage

Airway

Primary and secondary airway problems are at least as common in children as in adults. Airway compromise (e.g. croup, foreign body aspiration) is more common in very young children.

> Supporting videos can be found on the YouTube channel EM CapeTown or at:
> http://www.youtube.com/channel/UCDP5VfSnQ3AB1xgPbMUTniw_

Assessment

Look for abnormal recessions or retractions, or apnoea. Examine oropharynx for foreign body. Listen for noisy breathing or other indicators of upper airway obstruction (stridor, increased work of breathing, increased secretions, snoring). Consider clues to the cause (i.e. rash-anaphylaxis, foreign body etc.).

Intervention

Intervention progresses from simple to advanced as for adults. Equipment sizes and drug doses need to be adjusted for age and weight.

Breathing

Breathing problems are a much more common cause for decompensation and arrest in children than in adults.

Assessment

Assessment should include work of breathing, signs of respiratory distress, respiratory rate, tidal volume, airway and lung sounds, cyanosis, and pulse oximetry. Use age appropriate values when deciding if vital signs are normal or not.

> **Danger signs: A and B**
> - Tachypnoea or bradypnoea
> - Cyanosis
> - AMS
> - Abnormal breath sounds, particularly stridor or noisy breathing
> - Severe recession or retractions
> - Grunting and the use of accessory muscles
> - Alar flaring
> - Child holding tripod or sniffing position
> - General ill appearance

Intervention

Provide supplemental oxygen and other interventions (i.e. nebulisation) as per (📖 p. 8).

> **Airway and breathing: adjusting for age and weight**
> - Position: flexion due to a child's larger occiput may result in airway obstruction. Place a pillow or blanket roll under the shoulders
> - Face mask: choose a mask that covers the mouth and nose, starting from the bridge of the nose and ending between the bottom lip and chin
> - OPA: size from the middle of the mouth to the angle of the jaw
> - NPA: length – from the tip of the nose to the trachus of the ear. Diameter – about the width of the patient's little finger fingernail
> - Self inflating bag: three commonly available sizes: infant, child, adult. Limit use of the infant size to the neonatal period
> - Tidal volume: deliver enough volume to result in visible chest rise
> - Ventilation rate: 20–30 per minute for infants; 15–20 in older children
> - ETT (cuffed) size: (age / 4) + 3
> - ETT (uncuffed) size: (age / 4) + 4
> - ETT depth: newborn (tip to lip, in cm) 6 + wt (kg); older child (age / 2) + 12 cm
> - Induction: ketamine 1.5–2 mg/kg; etomidate 0.3 mg/kg
> - Paralytics: succinylcholine 1.5 mg/kg (up to 2 mg/kg in infants)

Circulation

The most common causes for shock and cardiovascular compromise in children in Africa are primary respiratory arrest, hypovolaemia (from diarrhoea), sepsis and congenital heart disease.

Assessment

Assess the presence of a pulse. If absent (or under 60 bpm in infants), continue to CPR at once. If present, assess

both cardiac function (HR, rhythm, BP, capillary refill, skin colour and temperature, pulses) and end organ perfusion (mental status, urine output, skin perfusion).

Intervention

Patients with cardiac arrest should be resuscitated as per the algorithm on p. 22 (Figure 2.1).

Two important differences from the adult algorithm are:

1. With two rescuers, a compression: ventilation ratio of 15:2 may be used. For single rescuer CPR 30:2 is the accepted ratio
2. Children require age/weight appropriate doses of drugs and defibrillation

> **Circulation: adjusting for age and weight**
>
> Use a paediatric resuscitation reference to calculate the dose of resuscitation drugs.
> - Adrenaline: 0.01 mg/kg IV or IO (0.1 ml/kg of a 1:10 000 solution) every 3–5 minutes during CPR
> - Defibrillation (energy dose): 4 J/kg

Disability

Rapid assessment of neurological function includes level of consciousness and basic brain and spine function.

Assessment

- Assess D by evaluating either AVPU or GCS, posture and pupil response
- Also look for obvious focal neurological deficit and possible convulsions. In young children, fontanelles may be bulging (raised ICP, meningitis) or sunken (dehydration)
- Testing and correction of glucose is critically important

Intervention

- Always test and correct glucose. Glucose: 0.5–1 g/kg IV/IO (5–10 ml/kg of a 10% dextrose solution)
- Stabilise spine as indicated. See Hypoglycaemia p. 402, RAP Seizure p. 74 and Altered mental status in children p. 34

Exposure

Assessment

Inspection of the entire patient is essential to identify injuries, rashes, petechiae or other clinical clues. Perform exam rapidly and prevent hypothermia. Check core temperature and cover patient once inspection is complete.

Intervention

See also Approach to the child with rash (p. 147) and Approach to purpura (p. 154) for life-threatening rashes. Patients with severe fever may warrant external cooling. Severely hypothermic patients should be actively warmed (p. 204). Cover to avoid hypothermia, which may occur rapidly in small children.

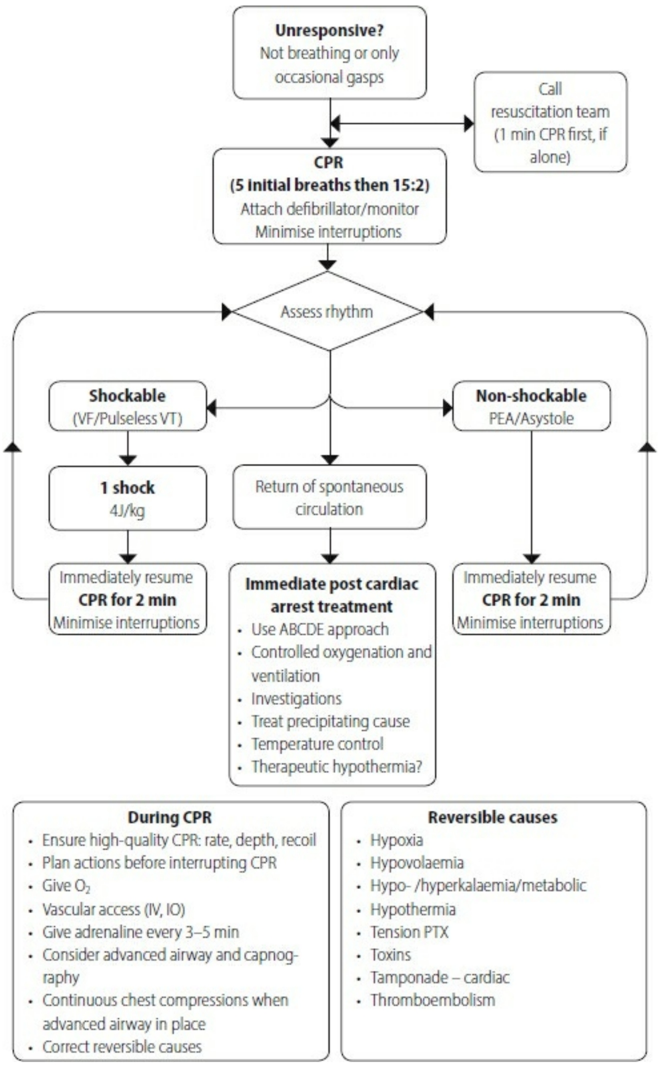


Figure 2.1 Paediatric advanced life support

3 Neonatal resuscitation: basic concepts and practice

Newborns undergo a radical change of physiology in the first few hours after birth. Most do this without problems and only < 1% require resuscitation. Resuscitation of the neonate differs significantly from that of adults and children.

Each step below should take no longer than 30 seconds.

Step 1: Initial evaluation (at birth)

Most newborns do not require resuscitation. Specifically consider:

- Term gestation?
- Breathing or crying?
- Good tone and activity?

If the answer to all three questions is **yes**, perform routine care:

Gently dry the baby and cover with a dry, warm blanket. Where possible, ensure a warm room (about 25°C), use warmed blankets/mattress, use a radiant heater, and place the baby in skin to skin contact with the mother, with exposed portions covered. Routine care includes providing essential neonatal interventions (such as conjunctivitis prophylaxis and vitamin K).

If the answer to **any** of the above questions is **no**, continue to step 2 and ask someone to call for assistance.

Step 2: Stimulate and reposition (first 30 seconds)

There are three key steps. If the infant does not breathe well or has a low heart rate (< 100 beats per min) after these steps, then resuscitation is required.

Warming and drying

Dry all term infants as above. For premature babies gently dab moisture off and consider placing the baby's body up to the neck in a clear plastic bag to reduce heat and fluid losses. Monitor temperature.

Stimulate

Gently rub the back or soles of the feet. Consider softly flicking the feet. Avoid slapping, shaking and other dangerous actions.

Position and clear the airway if necessary

Place a towel or blanket roll under the baby's shoulders to align airway (see Figure 3.1). Routine suctioning of the nose and mouth should not be performed (risk of bradycardia; no evidence of benefit). Consider careful suctioning of the airway if the baby displays signs of respiratory compromise.

Figure 3.1 Towel roll

When meconium present:
- If baby vigorous and crying: warm and dry, continue normal care
- If gasping, apnoea, HR < 100, poor tone: inspect orpharynx with laryngoscope and aspirate any meconium with large bore suction catheter
- If baby still unresponsive and INTUBATION POSSIBLE: intubate and aspirate trachea using tracheal tube as suction catheter
- If INTUBATION IMPOSSIBLE: clear oropharynx and start BVM

Step 3: Begin resuscitation (from 30–60 seconds)

- **Check breathing**. If the baby apnoeic or gasping, provide PPV by bag-valve-mask
- **Check pulse** by listening over precordium or palpating base of umbilicus. If pulse < 100, provide PPV
- **PPV**: use bag-valve-mask system or dedicated neonatal resuscitator device. Provide breaths at 40–60 per min with just enough volume to make chest rise. CAUTION: large volumes or pressures may result in PTX and irreversible lung damage
- **Oxygen**: begin with room air, use O_2 as indicated in the steps below

If available, attach SpO_2 monitoring to the right hand or wrist (pre-ductal, arterial blood).

Target saturation depends on minutes after birth: 60–65% at 1 min, 80–85% at 5 min, 85–95% at 10 min.

After 30 seconds PPV, re-check pulse. If pulse > 100 and baby breathing adequately with good colour and tone, continue post-resuscitative, regular newborn care. If not, further action depends on pulse and breathing efforts of baby.

Step 4: Continuing resuscitation (60–90 seconds)

Pulse 60–100: improve ventilation

- Readjust mask
- Reposition infant to open the airway
- Examine pharynx for obstruction and suction if necessary
- Consider airway adjuncts (LMA, OPA or intubation)

Pulse < 60: improve ventilation and begin compressions

Continue ventilation, but switch to 100% O_2. Start chest compressions – use two-hand encircling thumbs or one-hand, two finger technique (Figure 3.2). Compress 1/3 AP diameter of the chest with 3 compressions to 1 breath (3:1). Aim for 90 compressions and 30 breaths per min for total 30 cycles per min.

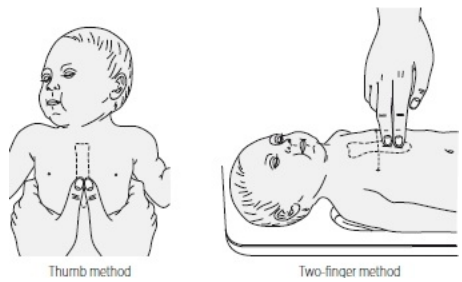


Figure 3.2 Thumb and two-finger method

Reassessment

After 30 seconds of performing the actions of step 4, re-check the pulse. If:

- > 100, check breathing and if adequate, discontinue O_2 (monitor response carefully) and continue post resuscitation care
- 60–100, continue and improve ventilation efforts
- < 60, go to step 5

Step 5: Administer adrenaline; continue CPR

If pulse remains < 60, continue quality CPR.

Give adrenaline 0.01 mg/kg (equal to 0.1 ml/kg of 1:10 000 solution) via umbilical vein catheter (📖 p. 822) (there is evidence that endotracheal route is ineffective). Administer every 3–5 min. If not intubated, consider now.

Other considerations

- **Hypovolaemia**: most newborns do not require a fluid bolus. Consider a 10 ml/kg isotonic crystalloid bolus in newborns with hypovolaemia (i.e. after bleeding from the cord)
- **Maternal opiates**: consider intubation and supportive care
- **PTX**: if breath sounds not present on one side and chest rise unequal, use needle decompression (📖 p. 636)
- **Glucose**: check and correct glucose. If glucose < 2.6 mmol/L, use 2.5 ml/kg of a 10% solution
- Currently no evidence supporting use of naloxone during neonatal resuscitation

Neonatal sepsis: in the case of maternal fever (≥ 38.5°C), newborn fever (≥ 38°C), prolonged rupture of membranes (> 12 hours), lethargic infant, or known maternal group B streptococcus, begin ampicillin 50 mg/kg IV/IM and gentamicin 4 mg/kg IV/IM (📖 p. 966).

- **Congenital heart disease**: if central cyanosis and murmur in an otherwise well-appearing infant, consider CCF and consult a specialist if available
- **Viability**: consider viability based on weight and age with locally available resources

- Dry the baby
- Remove any wet towels and cover
- Start the clock or note time
- Skin to skin

↓

Assess (tone), breathing and heart rate

↓

If gasping or not breathing:

- Open the airway
- Give 5 inflation breaths
- Consider SpO_2 monitoring
- Start with room air

CAUTION: Use minimum volume and pressure needed for chest rise. High risk of lung injury

↓

- Re-assess
- If no increase in heart rate, look for chest movement

↓

If chest not moving:

- Recheck head position
- Consider 2-person airway control an other airway manoeuvres
- Repeat inflation breaths
- Consider SpO_2 monitoring
- Look for a response

→

Acceptable pre-ductal SpO_2

2 min	60%
3 min	70%
4 min	80%
5 min	85%
10 min	90%

↓

- If no increase in heart rate, look for chest movement

↓

When chest is moving:

- If heart rate is not detectable or slow (< 60/min)
- Start chest compressions
- 3 compressions to each breath
- Add O_2 per text

↓

- Re-assess heart rate every 30 s
- If heart rate is not detectable or slow (< 60/min), consider venous access and drugs
- Add specifics on adrenaline per text

4 Volume resuscitation of children

Initial bolus to restore perfusion

See RAP Dehydration (p. 50) and evaluate for severe malnutrition (see SAM p. 394).

Give up to three serial **20 ml/kg IV/IO** boluses of NS or LR as needed for signs of poor perfusion. For mild to moderate dehydration, use enteral hydration if rapid administration tolerated.

Special considerations

- **Malaria or severe anaemia**: early transfusion and caution for haemodilution
- There remains controversy in the literature about whether **bolus fluids** may increase morbidity in very ill children who are not in shock, especially those with severe anaemia, but restoration of perfusion for shocked children is essential
- **Septic shock**: up to three boluses to restore perfusion. If >60 ml/kg required, consider blood transfusion
- Concern for **heart failure**: begin with 5–10 ml/kg aliquots and reassess frequently
- **Trauma**: divide boluses into 10 ml/kg aliquots with recheck in between
- **Burns**: see Parkland formula in Acute burn injury (p. 776)
- **Head injury** or recent surgery: avoid hypotonic IVF. Consider 0.9% NS in all age groups

Rehydration phase

Rapid rehydration (most children > 3 months)

Moderate to severe dehydration with no contraindications:

- ORS by NGT OR IV D5–½ NS with 20 mEq KCl* at 20 ml/kg/hr × 4h

Rapid rehydration contraindicated (<3 months or high risk)

Some children at high risk for fluid overload should be rehydrated over 24 hours rather than four hours.

Contraindications to rapid rehydration

Infants < 3 months
Severe acute malnutrition
Cardio-respiratory co-morbidity
Neurological co-morbidity
Hypernatraemia either by lab or exam (irritable, doughy-feeling skin, seizures)

High risk features above but shock resolved and no SAM

ORS by mouth or NGT preferred. If unable to take PO at rate described, use D5–½ NS with 20 mEq KCL*. Combine maintenance + rehydration + on-going losses and **administer IV/IO over 24 hours**.

Maintenance volume <3 months >3 months	 150 ml/kg/day divided over 24h 4:2:1 rule as below
Rehydration volume 5–10% dehydrated (moderate) =10% dehydrated (severe)	 50 ml/kg/d divided over 24h 100 ml/kg divided over 24h
Ongoing losses	Add 10 ml/kg for each loose stool or vomit

Reassess with Q4h checks. No other PO except breastfeeding for four hours. Reduce IVF and increase oral fluids as soon as possible. Serial blood glucose.

Maintenance phase (start after rehydration phase)

Rate

‘4, 2, 1 rule’ (Holliday-Segar Method):

- Give 4 ml/kg/hr for the first 10 kg of weight
- Add 2 ml/kg/hr for kilograms 11 through 20
- Add 1 ml/kg/hr for each additional kilogram

For example, a 12 kg patient would receive maintenance IV fluids at a rate of 44 ml/hr (the first 10 kg × 4 ml/hr = 40 ml/hr, and the additional 2 kg × 2 ml/hr = 4 ml/hr).

A 25 kg patient would receive maintenance IV fluids at a rate of 65 ml/hr (the first 10 kg × 4 ml/hr = 40 ml/hr, the second 10 kg × 2 ml/hr = 20 ml/hr, and the last 5 kg × 1 ml/hr).

Fluid choice

- Neonates/infants: D5% + ½ normal saline (D5% + ¼ NS may be considered in neonates, but can lead to iatrogenic hyponatraemia)
- Children: D5% + ½ normal saline
- Adolescents: D5% + normal saline
- Do not add K to fluids unless certain patient is producing urine

Potassium supplementation

- If paediatric patient is producing urine and has no known renal failure or hyperkalaemia, 20 mEq KCl per litre may be added to maintenance IVF

  **May substitute D5%–½ Darrows (½ DD), which contains ~17 mEq/L of K, for D5% –½ NS with 20 mEq KCl*

Part

3

Rapid assessment protocols

5 Abdominal pain in children

IMMEDIATE
0–5 minutes

HISTORY
- AMPLE history
- Onset, progression, duration
- Associated fever, vomiting (blood?), last bowel movement, diarrhoea
- Possibly pregnant?

PHYSICAL
- **A** Emesis? Aspiration risk (due to AMS)
- **B** Aspiration? Hypoxia, crepitations
- **C** Shock? ↓*BP, rapid weak pulses, cool peripheries, diaphoresis, tachycardia, pallor*
- **D** AMS (from shock?)

DIAGNOSTICS
- VS
- Rapid blood glucose
- O_2 sat

THERAPEUTICS
- Suction
- Lateral positioning
- O_2 ± bag ventilation
- Dextrose 10% (5 ml/kg) if RBG ≤ 3.5

EMERGENT
5–15 minutes

HISTORY
- Pain: onset, location, severity, progression, aggravating factors, constant?
- Distress with feeding or cardiac history?
- Occult trauma?
- Ingestion?

PHYSICAL
- Look for distension, ecchymoses, scars, jaundice
- Listen for bowel sounds
- Palpate for tenderness, guarding, masses/HSM, fluid wave
- Assess for dehydration
- Throat exam (strep associated with abdo pain)
- Respiratory (lower lobe pneumonia?)
- Cardiac (poor output → mesenteric ischaemia)
- Rash?

DIAGNOSTICS
- Bedside US: free fluid?
- Blood for crossmatch (if trauma or severe anaemia)

THERAPEUTICS
- Consider intubation if airway compromise
- Analgesia (morphine 0.1 mg/kg)
- NPO
- Consider NGT
- IV bolus (20 ml/kg)

URGENT
15–60 minutes

HISTORY
- Complete PMH, FH, SH
- System review
- Previous episodes
- Travel/camping
- Immunisations (rotavirus, hepatitis)

PHYSICAL
- Repeat abdo exam, complete exam, including f-up on:
  - » Additional patient complaints
  - » Findings from workup

DIAGNOSTICS
- CBC, blood grouping (if trauma or anaemia), electrolytes, liver enzymes, PT/PTT, lipase
- UA and urine pregnancy
- CXR and abdo XR, GI contrast study (volvulus)
- CT (if needed; US preferred in children)
- Endoscopy (PUD)

THERAPEUTICS
- Antibiotics for suspected infection
- Repeat IV bolus if hypotensive
- Consult surgery if peritoneal signs

PROMINENT FEATURES that would suggest … → RELEVANT CHAPTERS

PROMINENT FEATURES that would suggest …	RELEVANT CHAPTERS
Fever: • Appendicitis • Gastroenteritis • UTI • Typhoid • Lower lobe pneumonia • Streptococcal pharyngitis • Pelvic inflammatory disease • Cholecystitis	**RAP: Fever in child p. 62** Appendicitis p. 234 UTI in children p. 606 Acute pelvic pain p. 502 Pharyngitis and tonsillitis p. 178 Diarrhoea p. 242 Pneumonia in children p. 334
Severe persistent vomiting: • Mid-gut volvulus (especially infants <1 month), malrotation • Obstruction (incarcerated hernia, pyloric stenosis) • Toxin-mediated gastroenteritis • Acute hepatitis	Bowel obstruction p. 236 Vomiting p. 274 Hernias p. 253 Pyloric stenosis p. 260 Child with jaundice p. 262 Hepatitis p. 344
Bowel changes: • Toxic ingestions (decreased or increased bowel sounds) • Typhoid (may be associated with diarrhoea or constipation) • Intussusception (episodic pain and blood in stool) No stool: obstruction, constipation, perforation (e.g. PUD) with illeus, Hirschprung's Diarrhoea: • Hepatitis • Infectious gastroenteritis • Haemolytic-uraemic syndrome • Necrotising enterocolitis (premature/very small infant)	**RAP The crying or irritable child p. 48** Approach to the poisoned patient p. 652 Bowel obstruction p. 236 GIB in children p. 250 Child with jaundice p. 262 Hernia p. 253 Intussusception p. 258 Diarrhoea p. 242
High or epigastric pain: • Pancreatitis • Peptic ulcer disease (GIB) • Gallbladder disease • Basal pneumonia	GIB in children p. 250 Pancreatitis p. 270 Peptic ulcer disease p. 272 Pneumonia in children p. 334
Jaundice: • Hepatitis • Biliary obstruction/cholangitis • Metabolic disorders • Haemolytic crisis (sickle cell)	Hepatitis p. 344 Shistosomiasis p. 352 Gallbladder disease p. 244 Child with jaundice p. 262
Pain without significant tenderness: • Urolithiasis (colicky flank pain, haematuria, +/- fever) • Torsion (testicular, ovarian) • Mesenteric ischaemia (if poor cardiac output) • Hyperkalemia • DKA (polyuria, hyperglycaemia) • HSP	Urolithiasis p. 602 Mesenteric ischaemia p. 268 Testicular pain p. 614 Diabetic ketoacidosis p. 406 Approach to purpura p. 147
Hepatosplenomegaly: • Hepatic congestion (CCF, myocarditis, pericarditis) • Malaria • EBV • Lymphoma • Amoebic cyst or liver/spleen abscess • Early sickle cell disease	Malaria p. 326 Oncologic emergencies p. 298 Parasitic infection of the GI tract p. 354 Child with jaundice p. 262
Rash • HSP • Typhoid • Meningococcemia • Viral syndrome	Meningitis p. 330 Approach to purpura p. 154 Approach to the child with rash p. 147 Typhoid/cholera p. 346
Vaginal bleeding or discharge	Ectopic pregnancy p. 480 Miscarriage p. 480 Septic abortion p. 480 Acute pelvic pain p. 502
Trauma • Non-accidental trauma • Viscus rupture • Solid organ laceration	Paediatric resus p. 18 Approach to paediatric trauma p. 730 Approach to suspected child abuse p. 906

6 Abdominal pain in adults

IMMEDIATE
0–5 minutes

HISTORY
- AMPLE history
- Onset, progression, duration
- Associated fever, vomiting (blood?), last bowel movement, diarrhoea
- Difficulty breathing?
- Possibly pregnant?

PHYSICAL
- **A** Emesis? Aspiration risk (due to AMS)?
- **B** Aspiration? Increased work of breathing?
- **C** Shock? *Hypotension, rapid weak pulses, cool peripheries, diaphoresis, tachycardia, pallor*
- **D** AMS (from shock?)

DIAGNOSTICS
- VS
- O_2 sat
- Rapid blood glucose

THERAPEUTICS
- Suction
- Lateral decubitus position
- $O_2 \pm$ bag ventilation
- Dextrose 10% (500ml) or 50% 1 ampoule if RBG ≤ 3.5

EMERGENT
5–15 minutes

HISTORY
- Pain: onset, location, severity, progression, aggravating factors, constant, previous episodes?
- Fever?
- Vomiting (blood?), diarrhoea, last bowel movement
- Appetite, pain/vomiting with eating?
- Cardiac history?
- Liver history?
- Trauma?
- Ingestion?

PHYSICAL
- Respiratory (lower lobe pneumonia?)
- Cardiac (signs of CCF or atrial fib → MI, mesenteric ischemia, AAA)
- Look for abdominal distension, ecchymoses, scarring, jaundice
- Listen for bowel sounds
- Palpate for tenderness, guarding, masses, HSM, fluid wave
- Assess for dehydration

DIAGNOSTICS
- Bedside US: free fluid?
- Blood for crossmatch (if trauma or severe anaemia)
- 12-lead ECG (ischaemia, pericarditis)
- Urine pregnancy test

THERAPEUTICS
- Intubation or NIPPV if airway compromise
- IV bolus 1 L if hypotension or dehydration
- Analgesia (morphine 5 mg IV/IM)
- NPO

URGENT
15-60 minutes

HISTORY
- Complete PMH, FH, SH
- Systems review
- Travel/camping/rare food exposures

PHYSICAL
- Repeat abdo exam
- Complete exam, including pelvic exam for women, and follow-up on:
  - » Additional patient complaints
  - » Findings from workup

DIAGNOSTICS
- CBC, blood grouping (if trauma or anaemia), electrolytes, liver enzymes, PT/PTT, lipase
- UA
- Erect chest CXR and abdominal XR, GI contrast study (volvulus)
- CT
- Endoscopy (PUD)

THERAPEUTICS
- Antibiotics if suspected infection or surgical abdomen
- Repeat IV bolus if hypotensive and lungs clear
- Consider NGT
- Consider Foley catheter

PROMINENT FEATURES that would suggest …	RELEVANT CHAPTERS
If fever, consider: • Appendicitis, cholecystitis, diverticulitis, spontaneous bacterial peritonitis • UTI, intra-abdominal abscess, TTP-HUS, typhoid	Approach to the adult with fever p. 313 Approach to sepsis p. 318 Gallbladder disease p. 244 Appendicitis p. 234 Lower GIB p. 248 Approach to ascites p. 240 UTI in adults p. 604 Coagulopathies p. 288 Typhoid/cholera p. 346
If severe persistent vomiting, consider: • Obstruction (consider incarcerated hernia, volvulus, malignancy) • Ingestion/toxin • Gastroenteritis	Bowel obstruction p. 236 Hernias p. 344 Approach to acutely poisoned patient p.380 Vomiting p. 274 Diarrhoea p. 242
If vomiting blood or bloody stools, consider: • Blood in vomit: peptic ulcer disease, Mallory-Weiss tear, liver disease with esophagaeal varices, enteroinvasive gastroenteritis (fever), mesenteric ischaemia, malignancy	Upper GI bleeding p. 246 Peptic ulcer disease p. 272 Lower GI bleeding p. 248 Diarrhoea p. 242 Approach to the adult with abdominal mass p. 230
If high or epigastric pain, consider: • Pancreatitis, peptic ulcer disease, gallbladder disease, AAA, basal pneumonia	Pancreatitis p. 270 Peptic ulcer disease p. 272 Gallbladder disease p. 244 Abdominal aortic aneurysm p. 112
If jaundice, consider: • Hepatitis, biliary obstruction/cholangitis (RUQ pain, fever), pancreatic cancer	Jaundice adult p. 264 Hepatitis p. 344 Gallbladder disease p. 244
If pain without focal abdominal tenderness, consider: • Mesenteric ischaemia (if poor cardiac output, vascular history, atrial fib), AAA, urolithiasis (colicky pain, haematuria), Torsion (testicular, ovarian), hyperkalaemia, DKA	Mesenteric ischaemia p. 268 Abdominal aortic aneurysm p. 112 Urolithiasis p. 602 Testicular pain p. 614 Acute pelvic pain p. 502 Potassium disorder p. 426
If hepatomegaly and/or splenomegaly, consider: • Hepatic congestion (CCF, myocarditis, pericarditis), hepatitis, malaria, EBV, lymphoma/leukaemia, amoebic cyst or liver/spleen abscess	CCF p. 104 Hepatitis p. 344 Malaria p. 326 Approach to the adult with abdominal mass p. 230 Schistosomiasis p. 352
If polyuria, hyperglycaemia, consider: • Diabetic ketoacidosis	Diabetic ketoacidosis p. 406
Free fluid on US, consider: • Viscous rupture (due to trauma or late-stage appendicitis, diverticulitis, obstruction), solid organ laceration (blunt or penetrating trauma), ruptured ectopic pregnancy, ruptured AAA, spontaneous bacterial peritonitis (liver disease, fever), ascites	Adult resus p. 8 Penetrating abdominal trauma p. 748 Blunt abdominal trauma p. 746 Pelvic trauma p. 750 Acute pelvic pain p. 502 Aortic injury p. 744 Approach to ascites p. 240
Pregnancy, vaginal bleeding, or vaginal discharge, consider: • Ectopic pregnancy, PID (fever, discharge), placenta abruptio (acute onset bleeding, pain)	First trimester vaginal bleeding p. 480 Antepartum haemorrhage p. 486 Vaginal discharge p. 500 Acute pelvic pain p. 502 Abnormal uterine bleeding p. 478
DIB, consider: • Lower lobar pneumonia (fever), MI (chest pain, cardiac risk factors), pericarditis, myocarditis (fever, chest pain)	Adult resus p. 8 Pneumonia in adults p. 332 Myocarditis p. 122 Pericarditis p. 124

7 Altered mental status in children

IMMEDIATE — 0–5 minutes

HISTORY
- AMPLE history
- Trauma, seizures, ingestions?
- Recent neuro changes?

PHYSICAL
A Protection, secretions?
B ↑or ↓ rate, pattern
C Shock? *Hypotension, rapid weak pulses, cool peripheries, diaphoresis, tachycardia, pallor*
D AVPU, signs of ↑ICP (unequal pupils, posturing), focal neuro deficits

DIAGNOSTICS
- VS (hypo or severe hypertension? Cushing's triad?)
- O_2 sat
- Rapid blood glucose

THERAPEUTICS
- C-spine immobilisation if trauma
- O_2 ± bag ventilation
- IV and bolus (20 ml/ kg) if shock
- Elevate head of bed 30°
- Dextrose 10% (5 ml/kg) if RBG ≤ 3.5
- ICP *temporising* measures: ONLY if impending herniation AND neurosurgery imminent, consider mannitol, hypertonic saline, hyperventilation

EMERGENT — 5–15 minutes

HISTORY
- Onset, evolution, duration
- Associated: fever, headache, N/V/D, DIB, polyuria
- Recent illness/medications, DM?
- Exposure to tick/snake/spider

PHYSICAL
- Mental status
- Eyes: deviation, nystagmus, infection
- Pupils/fundi for ↑ICP
- Cranial nerves, motor (tone, reflex, symmetry), sensory, cerebellar
- Active seizures?
- Check gait

DIAGNOSTICS
- Repeat blood glucose if hypoglycaemic

THERAPEUTICS
- Consider intubation if severe ALoC or deteriorating
- Early antibiotics if meningitis concern
- Anti-pyretics
- Suspected ingestion: consider antidotes
- Repeat IV bolus if dehydrated; if evidence of severe anaemia and not shocked, hold IVF until transfusion

URGENT — 15–60 minutes

HISTORY
- Detailed history, birth history
- Milestones
- Change in baseline activity
- Any medications in home (e.g. oral hypoglycaemics, anticonvulsants)
- PMH: seizures? recent infection or immunisations, feeding problems/weight loss
- FH: early deaths? DM? Developmental problems?

PHYSICAL
- Complete exam
- Survey for non-accidental trauma
- Repeat exam for evolving neuro
- Occult infectious: ears, mouth, skin
- Tox: cherry red colour (carbon monoxide), decreased bowel sounds, diaphoresis

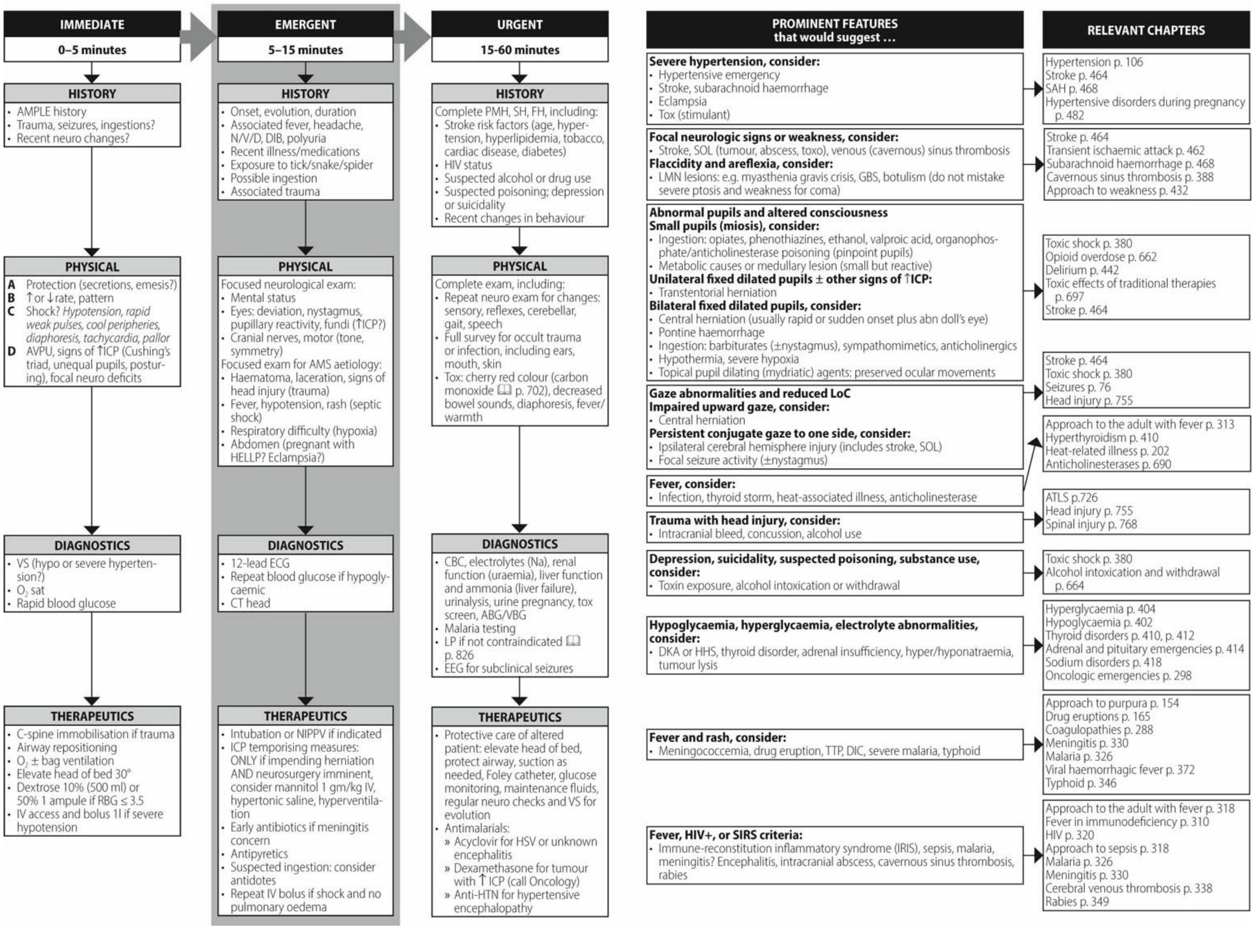
DIAGNOSTICS
- Blood culture, electrolytes (esp. Na), CBC, liver function, ammonia (congenital metabolic), urinalysis, tox screen, ABG/VBG
- LP with opening pressure if no contraindications
- LFT testing
- CT/MRI brain for mass, oedema, inflammation
- EEG for suspected subclinical seizures

THERAPEUTICS
- Antimalarials
- Acyclovir for HSV or unknown encephalitis
- Dexamethasone for tumour with ↑ICP (call Oncology)
- Anti HTN for hypertensive encephalopathy

PROMINENT FEATURES that would suggest … → RELEVANT CHAPTERS

PROMINENT FEATURES that would suggest …	RELEVANT CHAPTERS
Fever or other SIRS, consider: • Meningitis • Cerebral malaria • Seizure • HSV encephalitis • Intracerebral abscess (hx of ear infection, cyanotic cardiac lesion, penetrating head trauma, immunocompromise) • Thyroid disease • Intracerebral haemorrhage	**RAP Seizure in child p. 74** Meningitis, p. 330 Malaria p. 326 Approach to the child with fever, p. 316 Approach to the child with a rash p. 147 Purpura p. 154 Subarachnoid haemorrhage p. 468 Thyroid disorders p. 410 and p. 412 Fever in immunodeficiency p. 310
Focal neurological signs, consider: • Space occupying lesion (tumour, abscess, toxo) • Venous (cavernous) sinus thrombosis • Stroke (SCD, congenital cardiac shunt, hypercoagulable) • Active focal seizure • Post-ictal (Todd's paralysis)	**RAP Seizure in child p. 74** Approach to suspected child abuse p. 872 Sodium disorders p. 418 Hypertension p. 106 Hypoglycaemia p. 402 Sickle cell disease p. 282 Head injury p. 755 Cerebral venous thrombosis p. 388 Stroke p. 464
Vomiting ± diarrhoea, consider: • Dehydration • Electrolytes: Na, glucose, K • DKA • Toxin exposure, Reye's syndrome (if hx of aspirin use) • Inborn errors of metabolism	**RAP: Dehydration p. 50** Diarrhoea, p. 242 Vomiting p. 274 Child with jaundice, p. 262 Approach to SAM p. 394 Hypoglycaemia p. 402 Diabetic ketoacidosis p. 406 Sodium disorders p. 418 Post-infectious inflammatory disorders p. 382
Recent volume resuscitation (for V/D, DKA), consider: • Cerebral oedema due to hypotonic fluids • Electrolyte abnormalities	Sodium disorders p. 418 Volume resuscitation in children p. 27
Waxing and waning LoC, consider: • Intracerebral haemorrhage • Non-convulsive status epilepticus • Toxin exposure	**RAP: Seizure p. 74** Approach to suspected child abuse p. 872 Head injury p. 755 Toxic shock p. 380
Pupillary or gaze abnormalities **Small pupils (miosis), consider:** • Ingestion: opiates, phenothiazines, ethanol, valproic acid, organophosphate/anticholinesterase poisoning, intracranial lesion, ↑ICP **Bilateral fixed dilated pupils, consider:** • Central herniation (rapid onset with abnormal doll's eye) • Ingestion: barbiturates (nystagmus may also occur), sympathomimetics, anticholinergics - hypothermia, severe hypoxia • Topical pupil dilating (mydriatic) agents: preserved ocular movements **Persistent conjugate gaze to one side, consider:** • Ipsilateral cerebral hemisphere injury (includes stroke, SOL) • Ongoing seizure activity (± nystagmus) **Opthalmoplegia** • Botulinum • LMN: myasthenia gravis crisis, Miller-Fisher variant of GBS	**RAP: Weakness in children p. 80** **RAP: Seizures in children p. 74** Botulism p. 360 Delirium p. 442 Toxic shock p. 380 Toxic effects of traditional therapies, p. 697 Stroke p. 464 Approach to weakness p. 432 Head injury p. 755 Paediatric oncology presentations p. 304
Reduced LoC and abnormal motor **Dystonia or dyskinesia, consider:** • Ingestion: e.g. phenothiazines, phenytoin (+ nystagmus) • Inborn errors of metabolism • Encephalitis: brain stem encephalitis, encephalitis lethargica **Multifocal myoclonus, consider:** • Metabolic encephalopathy	**RAP: Weakness in children p. 80** Toxic shock p. 380 Approach to weakness p. 432

8 Altered mental status in adults



9 Anaphylaxis and angio-oedema

IMMEDIATE
0–5 minutes

HISTORY
- AMPLE history
- Known allergen exposure or angio-oedema?

PHYSICAL
A Airway oedema, stridor, vomit
B Wheezing, dyspnoea, stridor, hypoxia
C Shock? *Hypotension, weak pulses, cool periphery, diaphoresis, tachycardia, syncope*
D Lip, tongue, facial swelling, hypotonia

DIAGNOSTICS
- VS
- O_2 sat
- Random blood glucose

THERAPEUTICS
IM adrenaline injected into the anterior thigh:
- » 0.5 mg adult
- » 0.3 mg child
- » 0.01 mg/kg infant
- Airway repositioning: if obstructing tongue oedema (angio-oedema), patient supported upright
- 100% O_2 or bag mask ventilation
- 2 large-bore IVs. Place IO if no IV. First NS bolus 2l (or 20 ml/kg child)

EMERGENT
5–15 minutes

HISTORY
Voice change, sensation of throat closing or choking, drooling
Lip or tongue swelling?
Wheezing or DIB?
Dizziness or syncope?
Rash?
GI symptoms?
Prior episodes?

PHYSICAL
Repeat ABCs for response to adrenaline:
A Lip/tongue/uvular swelling improved? (Avoid suction or airway irritants!)
B Wheezing, dyspnoea, stridor, ↓air entry, ↑work of breathing, hypoxia
C *Signs of shock?* Hypotension, weak pulses, cool periphery, diaphoresis, tachycardia, syncope
D Expose patient. Hives, rash, itching, oedema? Remove any triggering allergen

DIAGNOSTICS
- Per suspected differential

THERAPEUTICS
Repeat adrenaline every 5–15 min as needed.
If hypotensive or shocked, repeat IV bolus every 15 min as needed until response
If DIB, salbutamol:
- 10 mg in 5 ml NS (adult)
- 5 mg in 3 ml NS (child)

If stridor or lip or mouth oedema, adrenaline 1:1000 nebulised:
- 0.5 ml/kg (max 5 ml)

If respiratory distress or hypotension/shock, consider adrenaline infusion. Mix 1 mg adrenaline in 1 litre saline (makes 1 mcg/ml solution:
- 3–10 mcg/min adult
- 0.1–0.3 mcg/kg/min child
- Titrate to response

If respiratory compromise and no response to adrenaline:
- Consider intubation. Prepare airway adjuncts and cricothyrotomy and consult anaesthesia

URGENT
15-60 minutes

HISTORY
Complete history including:
- Time and rapidity of onset
- Potential causes (see facing page)
- History of asthma?

PHYSICAL
Complete PE, including:
- Conjunctival oedema or injection
- Uvular oedema or erythema, tongue swelling, lip swelling (angio-oedema?)
- Pulmonary wheezes, prolonged expiratory phase, signs of obstruction
- Thorough skin exam for bites, stings, scratches, exposures, rashes, hives

DIAGNOSTICS
- Consider CXR for persistent wheeze
- Others per suspected differential

THERAPEUTICS
If persistent shock despite IVF and adrenaline, consider:
- Dopamine infusion 5–15 mcg/kg/min

Steroid: 🕮 Common emergency medications p. 959
- Hydrocortisone 200 mg IM/IV (or 5 mg/kg child)
- Methylprednisolone 125 mg IM/IV (adult or 1 mg/kg child)
- Dexamethasone 16 mg IM/IV (or 0.6 mg/kg child)

Antihistamines, e.g. chlorpheniramine (H1) and ranitidine (H2) (🕮 Common emergency medications p. 959)
- If known hereditary angio-oedema, FFP 10–15 ml/kg
- If on beta-blocking medication (propranolol, atenolol), glucagon 5 mg IV over 5 min (adult). Repeat as needed until response. Caution for emesis – administer over 5 min

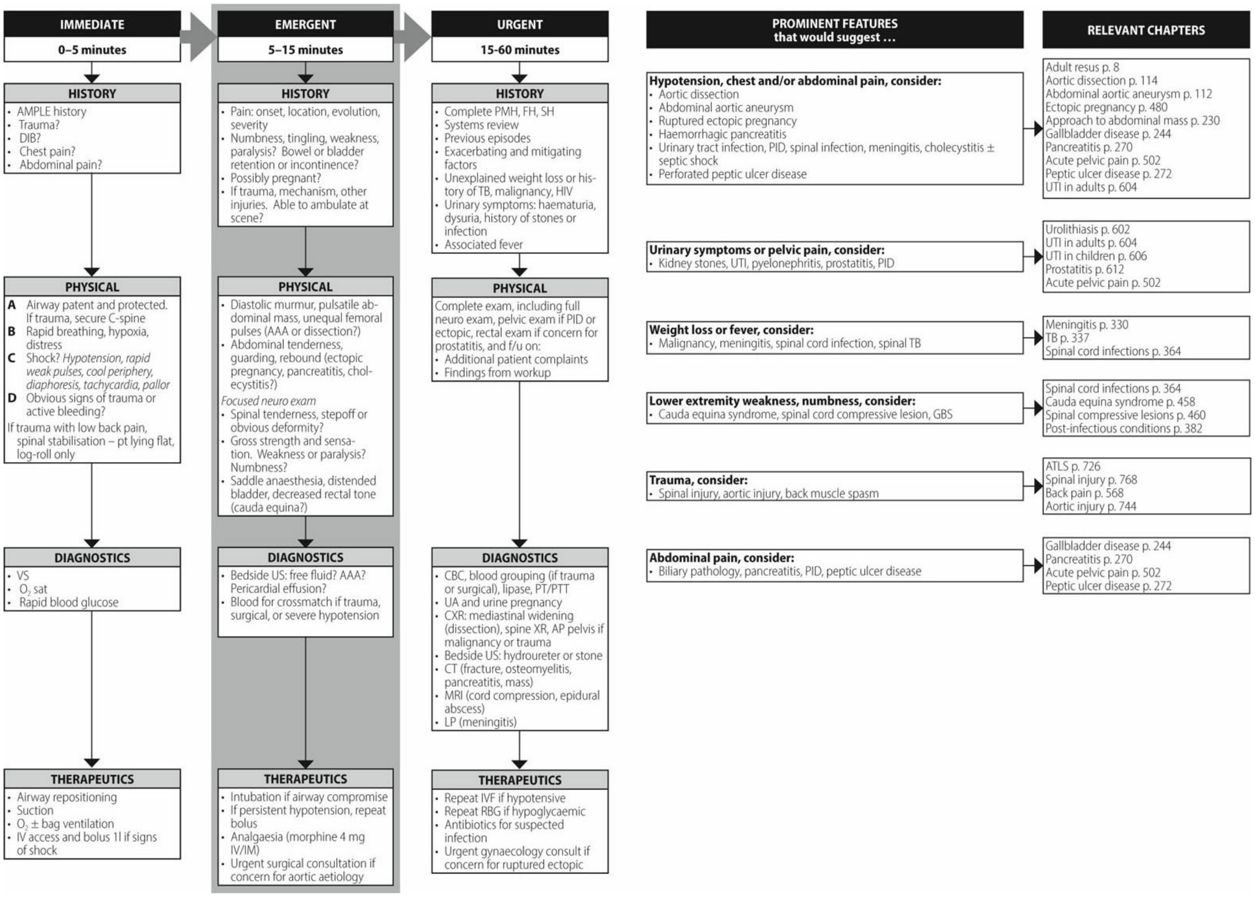
PROMINENT FEATURES that would suggest …	RELEVANT CHAPTERS
Airway oedema, stridor, consider: • Epiglottitis • Foreign body • Croup • Obstructing neck mass	Adult resus p. 8 Approach to stridor p. 172 Epiglottitis p. 176 Foreign body p. 184 Croup p. 174 Head and neck mass in adults p. 187 Head and neck mass in children p. 190
Wheezing, dyspnoea, hypoxia, consider: • Asthma • COPD exacerbation • Pulmonary oedema/CCF	Asthma p. 626 COPD p. 628 Dyspnoea p. 630 CCF p. 104
Fever, consider: • Epiglottitis, retropharyngeal abscess, peritonsillar abscess, bacterial pneumonia, SARS, AIDS-related pulmonary infection (pneumocystis, disseminated TB, fungal)	Epiglottitis p. 176 ENT infections p. 170 Pneumonia in adults, in children p. 332, p. 334 HIV p. 320 Influenza and SARS p. 340
Non-immunologic anaphylaxis (anaphylactoid reactions): • Transfusion reaction • Scombroid/ciguaterra • Iodine-based contrast agents	Transfusion reaction p. 292 Seafood toxins p. 722 Bee and ant stings, marine stings p. 218, p. 216

Definition of anaphylaxis (must meet criteria 1, 2, or 3). Reduced BP is ↓30% or SBP<90).
1. Rapid-onset illness with involvement of skin or mucosa AND EITHER:
   » Respiratory difficulty (wheezing, cough, stridor) OR ↓BP (or syncope)
2. Two or more of following within minutes–hours after exposure to a LIKELY allergen: involvement of skin or mucosa, respiratory compromise, ↓BP, persistent GI symptoms
3. ↓BP after exposure to a KNOWN allergen for that patient (minutes to several hours)

Common anaphylaxis triggers
- Foods: nuts, fruits (tomato, mango, banana, papaya), shellfish and fish, milk, egg, sesame, whey
- Venoms: insect bites, stings (bees, wasps)
- Medications, especially new or higher dose: aspirin, NSAID, sulfa, penicillin, estrogens, animal-derived serums (e.g. anti-venom)
- Exertion/exercise
- Latex

Common angio-oedema triggers:
- ACE inhibitors or angiotensin receptor blockers (ACEIs and ARBs)
- Oestrogens
- Calcium channel blockers
- Nonsteroidal antiinflammatory drugs (NSAIDs)
- Hereditary or acquired C1 inhibitor deficiency

10 Back pain in adults



11 Burns

IMMEDIATE
0–5 minutes

HISTORY
- AMPLE history
- Type (e.g. flame/liquid oil)
- Risk of other injuries: falls, explosions
- Risk of smoke inhalation: fire in enclosed space?

PHYSICAL
A Signs of inhalational injury (soot near mouth/nose, black sputum, stridor or hoarseness) → early intubation
B Tachypnoea, abnormal chest movement, circumferential chest wall burns, cyanosis
C Tachycardia, hypotension, evidence of haemorrhage
D AMS? Pupils, signs of obvious head injury
E Remove ALL clothing and jewelry (risk of continued burn, constriction). Rapid assessment of extent of burn (% body surface area per rule of 9s)

DIAGNOSTICS
- O_2 sat
- Cardiac monitor if possible
- Blood pressure
- Rapid blood glucose

THERAPEUTICS
- C-spine immobilisation
- Airway repositioning
- O_2 ± bag ventilation
- IV access preferably through unburned skin. IO if needed. 2 IV if > 30% burn
- NS IV bolus 20 ml/kg (child) or 1l (adult)

STOP BURN:
- Remove anything touching skin
- If burn < 3 hrs old: flowing clean tepid water over burn (avoid hypothermia)
- If victim of fire in enclosed space presents in arrest, empiric cyanide treatment

EMERGENT
5–15 minutes

HISTORY
- Time burn occured
- Specifics of agent causing burn
- Duration of contact
- Electrical shock?
- Associated trauma?
- Any IVF or analgesia already given, or topical treatment applied?

PHYSICAL
Assess burn
- % body surface area
- Depth
- Special areas involved
- Circumferential burns

Secondary survey
- Head to toe examination, particular attention to respiratory exam
- Presence of other injuries

DIAGNOSTICS
- Blood for crossmatch
- ABG (with carboxy- and met-Hgb level)
- US: (E)FAST if trauma
- ECG for all electrical burns and abnormal rhythm on palpation or monitor

THERAPEUTICS
- Intubation early if airway compromise anticipated or present
- Surgical airway/ needle cricothyroidotomy if unable to intubate
- Salbutamol for wheeze
- Further fluid boluses if hypotension and shock persisting
  - » Fluid resuscitation per Parkland Formula (📖 p. 776)
- In children: add dextrose-containing maintenance for burns > 10%
- Analgesia (intranasal, IV, oral)

URGENT
15–60 minutes

HISTORY
- Events surrounding incident
- Past medical history
- Social history
- Other persons injured?
- Consider non-accidental injury

PHYSICAL
- Repeat assessment of airway for any progressive symptoms (→ intubation)
- Monitor for hypothermia
- Evaluate for sepsis if subacute burns

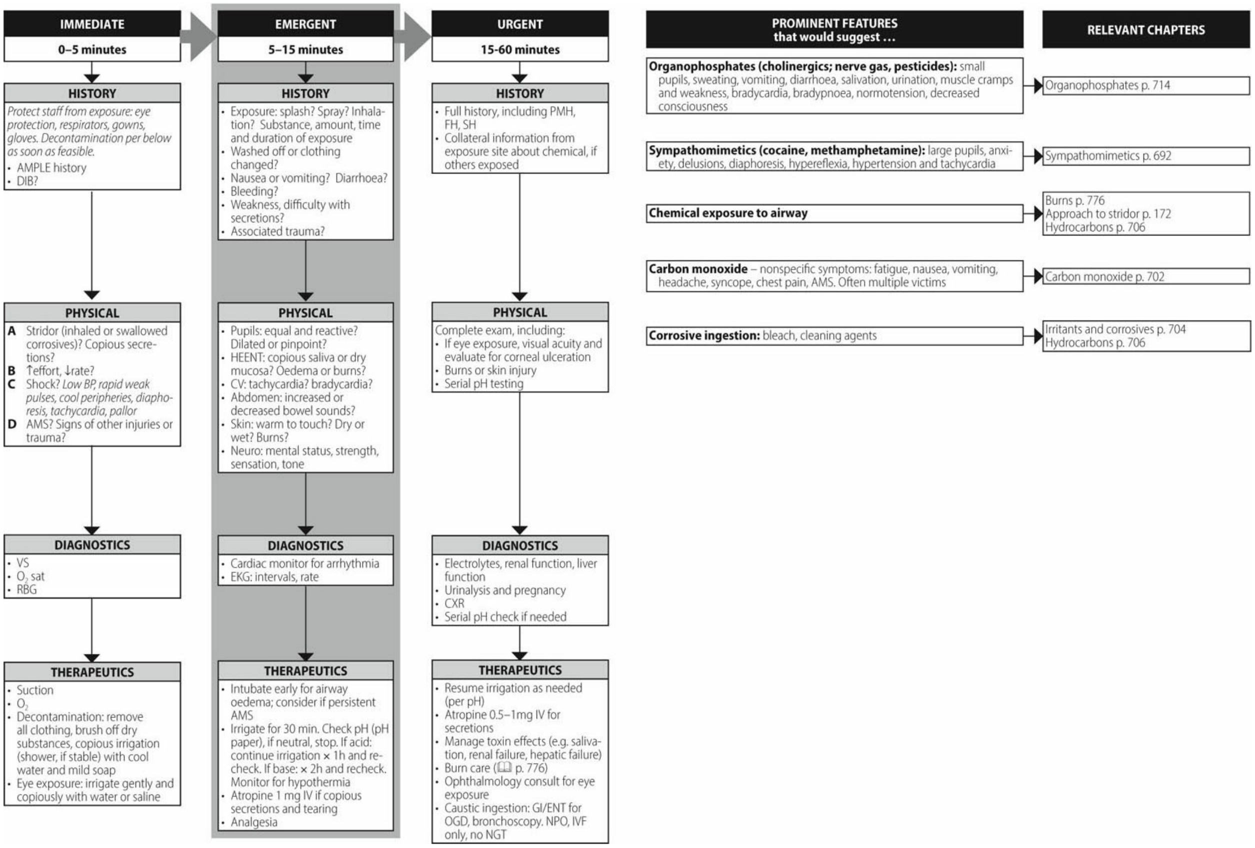
DIAGNOSTICS
- CXR
- CBC, renal function & electrolyte, coags
- UA and CK for all electrical burns (evidence of myoglobinemia/ rhabdomyolysis)
- Urine pregnancy
- Blood cultures if sepsis suspected
- CT as indicated for trauma (📖 Paed trauma p. 730)
- Monitor for pulmonary oedema and compartment syndrome with IVF

THERAPEUTICS
- Escharotomies (restrictive chest/ neck or circumferential limb burns) (risk of compartment syndrome)
- Chest tube for PTX
- Dress per burns chapter (📖 p. 776)
- Urinary catheter in burns > 20% /perineal burns. Monitor output
- Continue analgesia
- Tetanus toxoid
- Discussion with burn centre

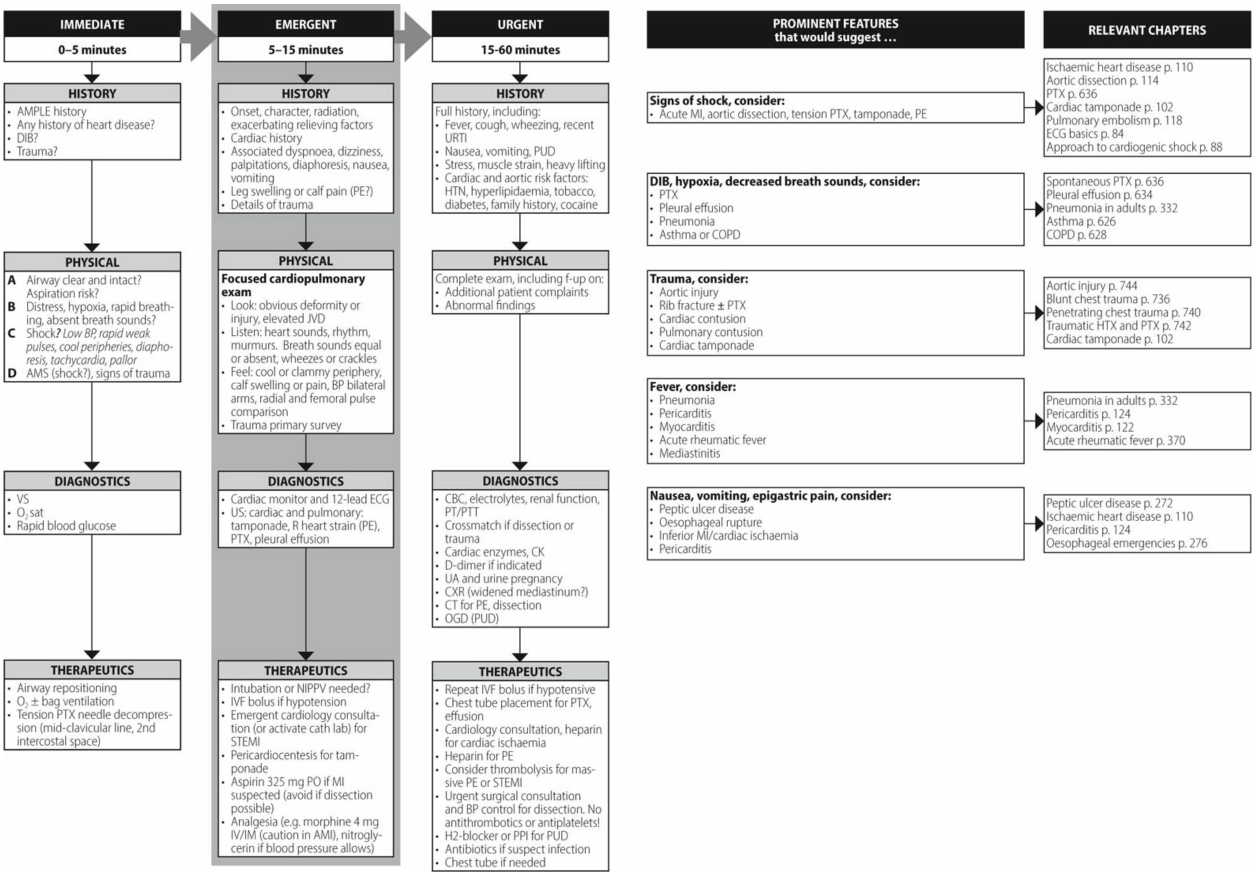
PROMINENT FEATURES that would suggest …	RELEVANT CHAPTERS
High risk for **airway burns:** • History of fire in confined space • Burns to face or neck • Singed hair/eyebrows • Hoarse voice • Carbonaceous sputum or deposits in and around mouth • Carboxyhaemoglobin > 10%	Unstable patient p. 6 Cricothyrotomy p. 862
High risk for **poly-trauma**: • History of explosion/jumping from building • AMS, PTX, hypotension, or fracture on initial presentation	Paed trauma p. 730 Blast injuries p. 722 PTX and HTX p. 742
• History of **electrical** burn (or found down with burns of unknown source) • History of **caustic chemical** exposure	Paed trauma p. 730 Electrical injuries p. 208 Rhabdomyolysis p. 617 Irritants and corrosives p. 704
AMS/LoC, headache, arrythmia, arrest, or tachypnoea (esp. with normal saturation), consider: • Carbon monoxide poisoning • Cyanide poisoning • Methaemaglobinaemia If testing unavailable: • Assume CO poisoning in any patient with neuro symptoms • In unconscious patient, evaluate for head injury and assume all 3 above conditions present • Clinical findings described in specific chapters	Complications of smoke inhalation p. 210 Methaemoglobinaemia p. 710 Carbon monoxide p. 702 Cyanide p. 700

CONSULT BURN CENTRE where available for:
- Burn ≥ 10% for age < 12 or >50
- Burn ≥ 30% TBSA (≥ 20% full thickness) in any patient
- Burns over joints or on hands, feet, face or perineum
- Burn + inhalation injury
- Significant chemical or electrical burns

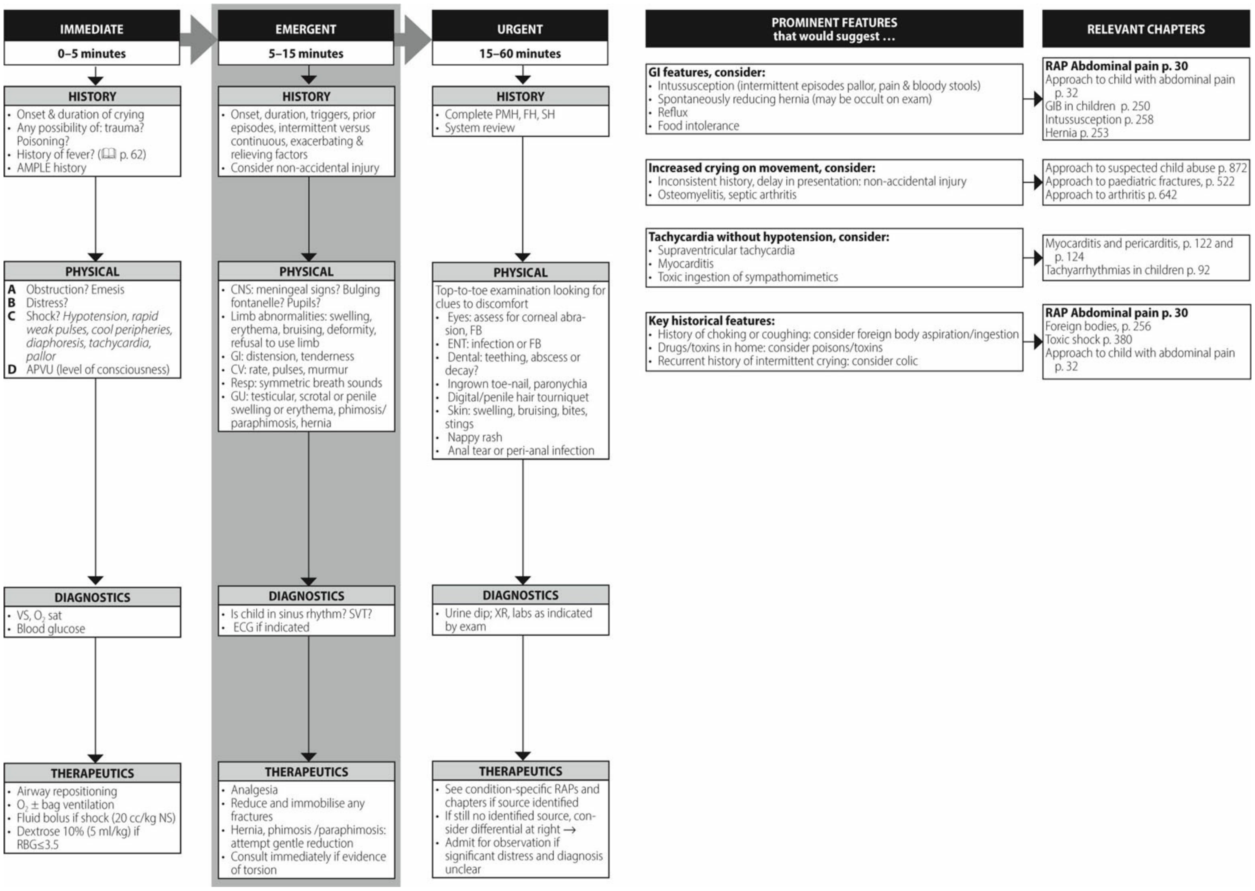
12 Chemical exposure



13 Chest pain in adults



14 The crying or irritable infant



15 Dehydration in children

IMMEDIATE
0–5 minutes

HISTORY
- AMPLE history
- PO and output: urine, tears, vomit, diarrhoea)
- Seizures, irritability, floppiness

PHYSICAL
A Emesis? Aspiration risk?
B Aspiration (crepitations)? Work of breathing?
C Shock? *Hypotension, rapid weak pulses, cool peripheries, diaphoresis, tachycardia, pallor*
D AMS? From shock, seizure, electrolytes

Signs of severe acute malnutrition (📖 p. 394)

DIAGNOSTICS
- VS
- Blood glucose
- O_2 sat
- Weigh child to guide rehydration

THERAPEUTICS
- O_2 ± bag ventilation
- Shock: IV bolus NS (20 ml per kg)
- Dextrose 10% (5 ml/kg) if RBG≤3.5
- If SAM, resuscitate per (📖 p. 394)

EMERGENT
5–15 minutes

HISTORY
- Stool: consistency, blood, mucus, quantity
- Vomiting: frequency, bile, blood
- Oral intake: type, amount, any ORS
- Urine output: consider DKA
- Possibility of toxin ingestion (incl traditional medicines)
- Similar illness in any contacts?

PHYSICAL
Nutritional status:
- Overweight (underestimates dehydration)
- Underweight (overestimates dehydration)

Degree of dehydration:
- MILD (< 5% deficit): thirsty
- MODERATE (5–10% deficit): very thirsty, irritable, dry mouth, sunken eyes
- SEVERE (> 10% deficit): very sunken eyes, lethargy, cap refill > 2s, ↓ skin turgor

DIAGNOSTICS
- CBC, electrolyte level, Na, K, ± urea
- ABG
- Blood culture if < 3 months
- Total protein, albumin if malnourished

THERAPEUTICS
Re-evaluate. If shock, repeat 20 ml/kg NS bolus. If perfusion not improved after 3 boluses, consider transfusion.

If perfusion improved, begin rehydration phase with rapid or high-risk protocol (📖 p. 27):
- Where available, non-sedating anti-emetics (ondansetron) if vomiting
- No feeds except breast milk
- NPO, IVF and NGT if obstruction suspected
- Treat fever (📖 p. 62)

URGENT
15–60 minutes

HISTORY
Full history including:
- Diet, heart, lung, GIT & endocrine
- Past medical/surgical history
- Immunisations

PHYSICAL
Complete examination:
- Look for subtle signs of sepsis
- Signs of endocrine/congenital disorders: dysmorphic features, abnormal genitalia

Reassess perfusion, hydration status:
- Re-weigh and compare to starting weight
- Volume retained and ongoing losses (stool, vomit, urine)

Complications:
- Assess respiratory status and level of consciousness (oedema?)

DIAGNOSTICS
- Electrolytes, ABG/VBG, total protein, albumin
- If <3 months: blood culture, UA/culture, consider LP
- CXR if indicated
- Stool studies if dysentery or high-risk, e.g. < 2y or immunocompromised
- Malaria testing
- HIV

THERAPEUTICS
- DKA protocol if relevant
- No antibiotics, unless signs of acute bacterial infection
- Avoid anti-diarrhoeal agents
- Ongoing management:
  - » Continue maintenance fluids

PROMINENT FEATURES that would suggest …	RELEVANT CHAPTERS
Fever, with: • Abdominal pain, headache, splenomegaly, consider: » Typhoid » Malaria • Failure to thrive, consider: » TB » Giardiasis » Malabsorption » Severe acute malnutrition » Coeliac disease	**RAP: Fever in child p. 64** Diarrhoea p. 242 Typhoid p. 346 Malaria p. 326 Approach to SAM p. 394
Prominent history of weight loss, consider: • DKA (excessive thirst, polyuria, lethargy) • TB (cough, dyspnoea, fever) • Cancer, tumour lysis syndrome • GI parasite • HIV	Metabolic and nutritional pp. 394–429 DKA p. 406 Paediatric oncology presentations p. 304 Parasitic infection of GI tract p. 354
Petechiae, purpura, high or low PTT, jaundice, consider: • Septicaemia • Hepatitis • HUS • DIC	Septicaemia p. 973 Hepatitis p. 344 Purpura p. 154 Acute kidney injury p. 594
Floppy, weak: • Electrolyte imbalance (low K or high Na) • Check other electrolytes, esp if convulsing (Ca, Mg, PO_4) • Adrenal crisis (hypotension, hypoglycaemia, ↑Na, ↓K) • Intoxication or envenomation	Metabolic and nutritional pp. 394–429 Hypokalaema p. 426 Hypernatraemia p. 418 Adrenal and pituitary emergencies p. 414
Prominent vomiting • Pyloric stenosis • Obstruction • Hepatitis	**RAP: Abdominal pain in children p. 30** Vomiting p. 274 Pyloric stenosis p. 260 Bowel obstruction p. 236 Hepatitis p. 344

16 Difficulty in breathing in children

IMMEDIATE
0–5 minutes

HISTORY
- AMPLE (asthma, prior pneumonia, cardiac?)
- Onset, duration, progression
- Fever or cough?
- Trauma, toxin

PHYSICAL

A Stridor/obstruction? Drooling, foreign body, angio-oedema, thermal/chemical burn
B DIB? RR, indrawing, exhaustion, cyanosis, dec or asymmetric air entry, tracheal shift (tension PTX), wheeze, chest wall trauma, nasal flare, grunting
C Shock? *low BP, rapid weak pulses, cool peripheries, diaphoresis, tachycardia, pallor*
D AMS? Restless, combative

DIAGNOSTICS
- VS
- SpO_2
- Cardiac monitor
- Rapid blood glucose

THERAPEUTICS
- Airway repositioning (protect C-spine if trauma)
- O_2 ± bag ventilation
- Complete obstruction: cricothyroidotomy
- Suspected epiglottitis: leave child upright, no intraoral exam, prepare OT
- Tension PTX: needle decompression (2nd intercostal space midclavicular)
- Bronchodilator (salbutamol neb)
- IV bolus (NS 20 ml/kg) if poor perfusion
- Angio-oedema/urticaria/severe bronchospasm: adrenaline (0.01 mg/kg) IV over 15 min or IM (max 0.3 mg)
- Dextrose 10% (5 ml/kg) if RBG ≤ 3.5

EMERGENT
5–15 minutes

HISTORY
- Onset, evolution, duration
- Associated: secretions, fever, voice change, feeding problems, noisy breathing, apnoea, emesis, polyuria
- Recent illness/medications

PHYSICAL
- Respiratory: chest wall movement, retractions, wheeze, grunting, crepitations, rhonchi
- CV: rate, HSM, JVP, perfusion, murmur
- Neuro: level of consciousness, seizures, meningismus, ↑ICP
- Skin: rash (urticaria, purpura, petechiae)

DIAGNOSTICS
- ABG (for O_2, CO_2, pH: acidaemia ⟶ tachypnoea)
- Ultrasound for PTX, consolidation, oedema, pleural/pericardial effusion, cardiac function, peritoneal fluid

THERAPEUTICS
- Intubation vs continued NIPPV
- Stridor: nebulised adrenaline (0.5 mg/kg in small amount NS, max for < 4 years = 2.5 mg, > 4 years 5 mg)
- Titrate slow IV adrenaline as needed for persistent symps
- Pericardiocentesis for tamponade

URGENT
15–60 minutes

HISTORY
- Complete PMH, FH, SH
- Detailed info on any prior episodes
- System review

PHYSICAL

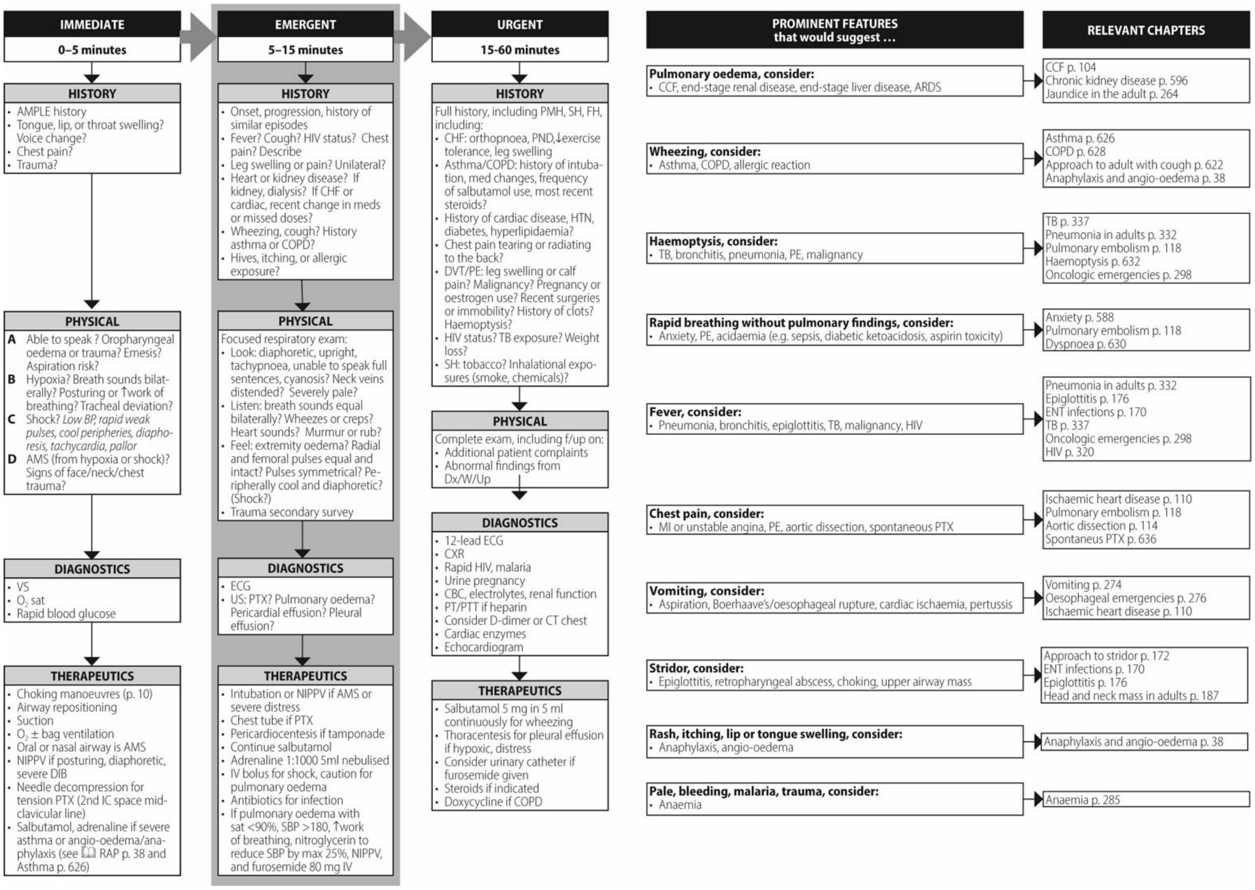
Full exam with: detailed ENT and cardiac exam

DIAGNOSTICS
- CXR, lateral neck XR
- CBC, blood grouping (if anaemia suspected), electrolytes, renal function, blood culture, CRP
- ECG if cardiac suspected
- Laryngoscopy/bronchoscopy if suspect foreign body

THERAPEUTICS
- Chest tube placement
- Croup, asthma: dexamethasone (0.15–0.6 mg/kg) or prednisolone 1–2 mg/kg
- Antibiotics
- Analgesia
- Continued bronchodilators, adrenaline as needed
- Treat pulmonary oedema (furosemide)

PROMINENT FEATURES that would suggest …	RELEVANT CHAPTERS
Stridor Foreign body, croup, airway burns, laryngomalacia, tumour obstructing airway, angioedema **Drooling** • Epiglottitis • Tracheitis • Retropharyngeal abscess	**RAP: Anaphylaxis and angio-oedema, p. 38** Croup p. 174 Epiglottitis p. 176 Approach to stridor p. 172 Paed resus p. 18 ENT infections p. 170 Complications of smoke inhalation p. 210
Choking history, severe retractions • Foreign body aspiration • Caustic inhalation or ingestion	Foreign bodies, p. 184 Cricothyrotomy p. 862 Approach to cough p. 624 Corrosive agents p. 704 Hydrocarbons p. 706
Distress with feeding • Laryngomalacia • Tracheal fistula • Reflux with aspiration	Vomiting p. 274 Crying or irritable child p. 48
Flushing, itching, urticaria • Allergic reaction ⟶ anaphylaxis	**RAP: Anaphylaxis p. 38** Approach to rash p. 147
Wheeze • Asthma, anaphylaxis • Viral infections, pneumonia, bronchiolitis • Reflux disease • Tracheomalacia • Paraffin or other toxic inhalation/ingestion • Pulmonary congestion/cardiac failure • FB aspiration	**RAP: Anaphylaxis p. 38** Pneumonia in children p. 334 Asthma p. 626 Corrosive agents p. 704 Hydrocarbons p. 706
Fever with cough • PNA (focal crepitations or dec breath sounds), viral infections, bronchiolitis, TB • Consider ARDS in septic patients	Pneumonia in children p. 334 TB p. 337 Approach to cough p. 624 Approach to the patient with sepsis p. 318 Reading the CXR p. 950
Decreased breath sounds • Bases ± crepitations: pleural effusion, pulmonary oedema • Focal lobar: PNA • Unilateral: PTX • Diffuse: severe asthma, obstruction	Pneumonia in children p. 334 TB p. 337 Approach to cough p. 624 Reading the CXR p. 950 Spontaneous PTX p. 636 PTX and HTX p. 742 Pleural effusion p. 634 Chest tube insertion p. 838
Cyanosis not correcting with O_2, or tachycardia out of proportion to respiratory difficulty, difficulty with feeding⟶ cardiac cause	Cardiogenic shock p. 88 CCF p. 104 Congenital heart disease p. 126
Oedema • Renal failure, nephrotic syndrome • Liver failure (infectious), toxins • Heart failure	**RAP: Oedema p. 56** Congenital cardiac disease p. 126 Kidney disease p. 596 Ascites p. 240
• **If weak cough, paradoxical rib cage movement** with inspiration, consider respiratory muscle weakness. • If suspicion of ingestion and absence of cardio-respiratory pathology, consider POISONING.	**RAP: Weakness p. 80** GBS, botulism, approach to weakness Toxic shock p. 380
Clear on auscultation, consider • Hyperpnoea/tachypnoea, metabolic cause (DKA, tox, other acidosis) • Hypoxia with clear lungs: PE	Diabetic ketoacidosis p. 406 Salicylates p. 658 Dehydration p. 50 PE p. 118 Pulmonary hypertension p. 108

17 Difficulty in breathing in adults



18 Oedema in children

IMMEDIATE
0–5 minutes

HISTORY
- AMPLE history
- DIB or rapid breathing?
- Onset, duration of swelling.
- Decreased or bloody urine?

PHYSICAL
- **A** Protected and intact?
- **B** Hypoxia? DIB?
- **C** Shock? ↓*BP, rapid weak pulses, cool peripheries, diaphoresis, tachycardia, pallor*
- **D** AMS? From shock, hypoxia, encephalopathy, electrolyte changes
- Check for severe acute malnutrition (p. 394)

DIAGNOSTICS
- O_2 sat
- VS
- RBG

THERAPEUTICS
- Upright positioning if respiratory distress
- O_2 ± bag ventilation
- If shock, assess pulmonary oedema (crepitations, hypoxia) and severe anaemia. If none, IV and 10 cc/ml fluid bolus
- Dextrose 10% (5 ml/kg) if RBG ≤ 3.5

EMERGENT
5–15 minutes

HISTORY
- Oedema: onset, progression, duration
- DIB? Worse lying down?
- Fever?
- Decreased urine? Red or brown colour?
- Cardiac, liver, or renal history?

PHYSICAL
- Respiratory: creps (pulmonary oedema)?
- Cardiac: elevated neck veins, displaced apical beat, murmurs or gallops (CCF)?
- Abd: palpable liver, distension, ascites?
- Skin: colour change, especialy in skin folds, groin, or ulcers (malnutrition)? Rashes, especially on legs (HSP)? Pale palms/soles (anaemia)? Jaundice?
- Pitting or nonpitting?

DIAGNOSTICS
- ECG
- Urine dip for protein, Hgb, bili

THERAPEUTICS
- If pulmonary oedema: NIPPV, consider intubation
- Furosemide 1–2 mg/kg IV
- If shock and no pulmonary oedema, repeat bolus 10 ml/kg. If shock with pulmonary oedema, PPV and consider IVF and furosemide together
- If renal, check ECG for ↑K (loss of P waves, wide QRS, bradycardia, peaked T) (p. 426) and treat: Ca, insulin/dextrose, salbutamol nebulisation. (Caution with Ca if on digoxin.)

URGENT
15–60 minutes

HISTORY
Full history, including:
- PMH: recent URTI or diarrhoeal illness, rash
- Headaches, fatigue, cold feeling, dry skin (thryoid)?
- Jaundice, pale stools (liver failure)?
- Medication history
- Nutrition: protein, fluid intake
- Immunisation history and growth chart

PHYSICAL
Complete exam, including:
- Thyroid: goitre? Decreased reflexes? Puffy face?
- GU: scrotal or labial oedema, skin breakdown? Abnormal genitalia?
- Evaluate height for age (short stature: malnutrition v hypothyroid)

DIAGNOSTICS
- CBC and smear, electrolytes, renal and hepatic function, protein, albumin, ABG/VBG, HIV, PT/PTT, TSH, free T4
- UA, urine electrolytes
- CXR
- If fever, blood, urine, ascites fluid cultures, malaria RDT/smear, stool culture (E. coli → HUS)
- Complement levels, strep antibodies

THERAPEUTICS
- Foley catheter
- CCF with persistent shock, consider vasopressors, inotropes
- Therapeutic tap for effusion or ascites causing DIB
- Nephrology for dialysis for acute kidney injury with AMS, ↑K, severe pulmonary oedema, acidosis.
- For severe hypertension, lower BP by no more than 20% (p. 106)

PROMINENT FEATURES that would suggest …	RELEVANT CHAPTERS
Known CCF, enlarged heart, gallop: • Congenital heart disease • Rheumatic heart disease, endomyocardial fibrosis • Infectious heart disease (myocarditis, pericarditis, endocarditis with valvular incompetence • Pulmonary hypertension • Fluid overload, e.g. iatrogenic, acute renal failure, liver failure	CCF p. 104 Congenital heart disease p. 126 Approach to cardiogenic shock p. 88 Pulmonary hypertension p. 108 Mitral valve disease p. 95 Myocarditis p. 122 Pericarditis p. 124 Endocarditis p. 100 Cardiomyopathies p. 120
• Urinary symptoms, hypertension, and/or rash, consider: • Acute renal failure • Acute post-streptococcal glomerulonephritis • Nephrotic syndrome • Haemolytic uremic syndrome • Henoch-Schönlein purpura	Acute kidney injury p. 595 Approach to the child with rash p. 147 Haematuria p. 599 Post-infectious conditions p. 382
Ascites, hepatomegaly, liver disease, consider: • Kwashiorkor • Cirrhosis • Abdominal TB • CCF • Acute or chronic renal disease	Approach to ascites p. 240 Approach to SAM p. 394 Jaundice in children p. 262 Hepatitis p. 344 TB p. 337 CCF p. 104 Acute kidney injury p. 595
Lymphedema or nonpitting oedema, consider: • Filariasis • Malignancy • Autoimmune disease (JIA, Crohn's, sarcoid) • Hypothyroidism	***Filariasis/Leischmaniasis*** Paediatric oncologic presentations p. 304 Hypothyroidism p. 412
Severe anaemia, consider: • Malaria • Malnutrition • Parasitic infection • Chronic kidney disease • End-stage liver disease • Sickle cell disease • Haematologic malignancy or aplastic anaemia • Gastrointestinal bleeding	Anaemia p. 285 Malaria p. 326 Approach to SAM p. 394 Coagulopathies p. 288 Acute kidney injury p. 595 Jaundice in children p. 262 Parasitic infection of the GI tract p. 354 Sickle cell disease p. 282 Paediatric oncologic presentations p. 304 GIB in children p. 250
Fever, consider: • Acute rheumatic fever • Myocarditis • Constrictive pericarditis • Endocarditis with valvular incompetence • Malaria • Haemolytic uremic syndrome • Henoch-Schönlein purpura	Approach to the child with fever p. 316 Myocarditis p. 122 Pericarditis p. 124 Endocarditis p. 100 Acute rheumatic fever p. 370 Malaria p. 326 Haematuria p. 599 Post-infectious conditions p. 382

19 Oedema in adults

IMMEDIATE
0–5 minutes

HISTORY
- AMPLE history
- DIB?
- Chest pain?
- Known CCF, liver or renal patient?

PHYSICAL
- **A** Aspiration risk?
- **B** Distress? *Hypoxia, rapid breathing, crepitations*
- **C** Shock? *Hypotension, rapid weak pulses, cool periphery, diaphoresis, tachycardia, pallor*
- **D** AMS (from shock?)

DIAGNOSTICS
- VS
- O_2 sat
- Rapid blood glucose

THERAPEUTICS
- Upright position (if orthopnea)
- NIPPV if respiratory distress
- $O_2 \pm$ bag ventilation
- If patient in shock with pulmonary oedema (crepitations, hypoxia), PPV with resuscitation via cautious 250 cc boluses
- Dextrose 50% 1 ampoule if RBG <3.5

EMERGENT
5–15 minutes

HISTORY
- Onset of oedema, progression, bilateral or unilateral
- DIB if supine, DIB or chest pain with exertion
- Possibly pregnant?

PHYSICAL

Focused exam for pulmonary oedema:
- Crepitations or decreased breath sounds
- Hypoxia
- DIB, ↑work of breathing?

DIAGNOSTICS
- 12-lead ECG (ischaemia?)

THERAPEUTICS
- If hypoxic, pulmonary crepitations, and SBP > 180 (→ flash pulmonary oedema), see 📖 DIB p. 54
- If persistent ↓BP with NO pulmonary oedema, repeat bolus
- Consider early pressors/inotropes if ↓BP with pulmonary oedema
- Treat ↑K (📖 p. 426)

URGENT
15–60 minutes

HISTORY
- Full history, including:
- CCF, ischaemia, HTN, diabetes, murmur
- Liver: hepatitis, alcohol
- Renal: urine output, haematuria?
- Hypothyroid signs?
- Nutrition (protein deficiency?)
- Medications

PHYSICAL

Complete exam, including:
- Thyroid: goitre or mass? Skin/hair changes?
- CCF: tachycardia? Irregular? Murmur? Displaced PMI?
- Liver: ascites? Jaundice? Portal HTN?
- Pregnancy
- Oedema: unilateral (DVT) or bilateral? Level?

DIAGNOSTICS
- CBC, electrolytes, renal function, liver function, troponin, ABG/VBG
- UA and urine pregnancy
- TSH (hypothyroidism)
- CXR or US for pulmonary oedema
- Renal US (kidney disease)

THERAPEUTICS
- Place Foley catheter
- If BP allows, furosemide 40–80 mg IV
- If renal patient with hypoxia, acidosis, consider dialysis
- If large pleural effusions or ascites with DIB and hypoxia, therapeutic thoracentesis or paracentesis

PROMINENT FEATURES that would suggest …	RELEVANT CHAPTERS
Hypotension or shock, consider: • Cardiogenic shock • Massive PE • ↑K due to renal failure	Adult resus p. 8 Approach to cardiogenic shock p. 88 Pulmonary embolism p. 118 Potassium disorders p. 426
Chest pain, DIB, consider: • PE • CCF: cardiomyopathy, myocarditis, MI, pulmonary HTN, congenital heart disease	**RAP: DIB p. 54** Pulmonary embolism p. 118 CCF p. 104 Cardiac tamponade p. 102 Congenital heart disease p. 126 Pulmonary hypertension p. 108 Cardiomyopathies p. 120 Ischaemic heart diseases p. 110 Myocarditis p. 122
Anasarca, consider: • CCF • Liver disease • Renal disease • Hypothyroidism	CCF p. 104 Approach to ascites p. 240 Chronic renal disease p. 596 Hypothyroidism p. 412
Bilateral lower extremity swelling, consider: • Bilateral DVT, potentially due to malignancy • Filariasis • IVC compression by abdominal mass • Pregnancy • Drugs (amlodipine) • Dependent or heat oedema • Anaemia • CCF	DVT p. 116 Approach to the adult with abdominal mass p. 230 Hypertensive disorders of pregnancy p. 482 Anaemia p. 285 CCF p. 104
Unilateral lower extremity swelling, consider: • DVT • Filariasis	DVT p. 116

20 Emergency delivery

IMMEDIATE
0–5 minutes

HISTORY
- Parity, gravidity, gestational age
- Complications in this or prior pregnancy?
- Contractions, onset and frequency? Rupture of membranes (colour, smell, quantity of fluid)
- PV bleeding
- LoC or seizures?

PHYSICAL
- ABCD and VS
- Timing of contractions
- Pallor or jaundice
- Abdominal tenderness, peritoneal signs
- Signs of imminent delivery (gaping of vulva)

DIAGNOSTICS
- O_2 sat
- Rapid blood glucose

THERAPEUTICS
If hypoxic or respiratory distress:
- Airway repositioning
- O_2 ± bag-valve mask ventilation
- 2 large bore IV and 2l bolus if hypotensive
- Left lateral position until delivery
- Dextrose 10% (500 ml) or 50% 1 ampoule if RBG ≤ 3.5
- If fetus crowning through the vulva, attend 2nd stage
- If breech presentation, call OB and see breech (📖 p. 493)

EMERGENT
5–15 minutes

HISTORY
- Risk factors for complicated pregnancy e.g. HTN, diabetes
- Past obstetric history
- Recent illness or sick contacts

PHYSICAL
- **Obstetric**: uterine size, fetal presentation, uterine contraction, FHR
- **Vaginal** (if no APH): cervical dilatation, presentation and station, presence of umbilical cord, drainage of fluid

DIAGNOSTICS
- Bedside US for fetal HR and activity

THERAPEUTICS
Consult OB immediately for: pre-term, non-vertex, cord prolapse, fetal distress, haemmorrhage, pre/eclampsia, shoulder dystocia
- If pre-term labour, consider tocolytics per OB consultation
- For cord prolapse: mother in knee-chest position and transfer to theatre with upward pressure on presenting fetal part
- If fetal distress, L lateral position, O_2, consider assisted delivery (e.g. forceps) or C-section
- APH/PPH: cross-match 6 units
- Mg sulphate for seizures or preeclampsia. Labetolol or hydralazine for HTN (📖 p. 482)

URGENT
15-60 minutes

HISTORY
Antenatal history: pregnancy baseline Hb, blood group and immunisations, HIV status

PHYSICAL
Complete exam, incl:
- Cardiac: evidence of failure?
- Any evidence of infection?

DIAGNOSTICS
- Hgb, blood group and Rh status
- Urinalysis
- HIV status

THERAPEUTICS
- If febrile, IV antibiotics to cover chorioamnionitis or pyelonephritis
- Continue IVF as needed

PROMINENT FEATURES that would suggest … → RELEVANT CHAPTERS

Painful, rhythmic uterine contraction leading to cervical dilatation
Normal labour:
- 1st stage: onset of labour to full cervical dilatation (10 cm)
- 2nd stage: delivery of the fetus
- 3rd stage: delivery of the placenta

→ Management of labour p. 490

AMS or seizure:
- Preeclampsia: HTN (BP>140/90 mmhg) AND proteinuria or non-dependent oedema at > 20 weeks gestation
- Eclampsia: seizures or coma in women with preeclampsia
- CNS infection

→ Hypertensive disorders of pregnancy p. 482
Meningitis p. 330
Approach to adult with fever p. 64

Persistent vaginal bleeding:
- **APH**: vaginal bleeding after 28 weeks gestation until delivery of the fetus. Consider placental abruption or placenta praevia
- **PPH**: excessive bleeding (>500 ml) after delivery of the fetus. Consider uterine atony and retained placental tissue

→ Vaginal bleeding in pregnancy p. 480
Antepartum haemmorrhage p. 486

Fever:
- Pyelonephritis
- Chorioamnionitis, endometritis
- Aspiration

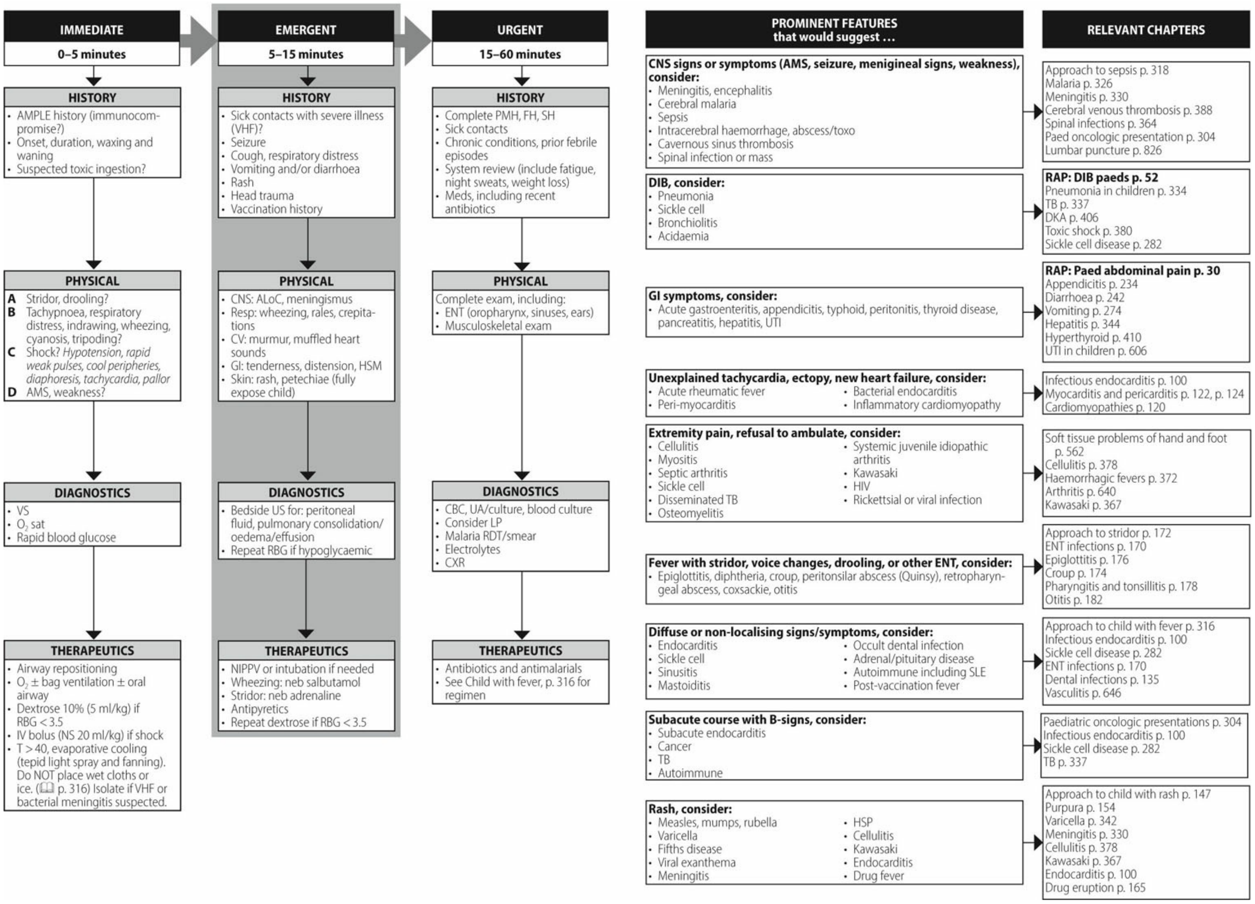
→ Urinary tract infection in adults p. 604
Adult with fever p. 64

Prolonged labor (or identified non-vertex presentation):
- Shoulder dystocia
- Cephalo-pelvic disproportion
- Breech or footling breech

→ Obstructed labour p. 485
Breech p. 493
Shoulder dystocia p. 496

See also Neonatal resuscitation p. 23

21 Fever in children



22 Fever in adults

IMMEDIATE
0–5 minutes

HISTORY
- AMPLE history
- Onset, duration, waxing and waning
- Toxic or environmental exposure?
- Rash?

PHYSICAL
A Airway patent/protected? Stridor, voice changes
B DIB or rapid breathing, hypoxia?
C Shock? *Low BP, rapid weak pulses, cool peripheries, diaphoresis, tachycardia, pallor*
D AMS? Signs of increased ICP (Cushing's triad, unequal pupils, posturing)?

Isolation if haemorrhagic fever or bacterial meningitis suspected

DIAGNOSTICS
- VS
- O_2 sat
- Rapid blood glucose

THERAPEUTICS
- Airway repositioning
- O_2 ± bag ventilation
- IV and 2L NS for shock
- If suspected ↑ICP, elevate HOB 30°
- T > 40, evaporative cooling (light spray with tepid water and fanning). Do NOT place wet cloths or ice

EMERGENT
5–15 minutes

HISTORY
Fever duration, timing:
- Head, neck, back pain
- Cough, difficulty breathing, wheeze
- Vomiting, diarrhoea
- Urinary frequency, pain, vaginal discharge
- Skin lesion
- Immune: HIV, malignancy, sickle cell, steroids, diabetes, alcoholism
- Any thyroid disease (thyroid storm?)
- Recent exertion or exposure to heat (heat-related illness?)

PHYSICAL
Brief exam for source:
- HEENT: posterior pharynx, thyroid, lymph nodes, nuchal rigidity
- CV: murmur, evidence of endocarditis?
- Pulm: crepitations, hypoxia (pneumonia?)
- Abd: distension, tenderness, guarding, hepatosplenomegaly
- FULL skin: jaundice, anaemia, rash, infection
- Skin very hot and dry? (Consider heat stroke or anticholinergic)

DIAGNOSTICS
- Rapid malaria
- Rapid HIV

THERAPEUTICS
- Consider intubation or NIPPV if severe respiratory distress
- Repeat IV bolus if persistent hypotension
- Antipyretics (paracetamol 1 gm PO/PR)

URGENT
15-60 minutes

HISTORY
- Complete history, including PMH, SH, FH
- Medications, including herbals or antimalarials
- Any prior testing?
- Weight loss, tremors, palpitations, diarrhoea, skin or hair changes
- If HIV+, on ARVs? When begun? (IRIS?)
- Recent surgeries, procedures, or trauma?
- Toxic exposure, illicit drugs, poisoning?
- Haemorrhagic: blood in vomit or stool, lips and gums, petechiae, vaginal bleeding

PHYSICAL
Complete exam, including:
- Pelvic exam for women
- Scrotal and GU exam for men

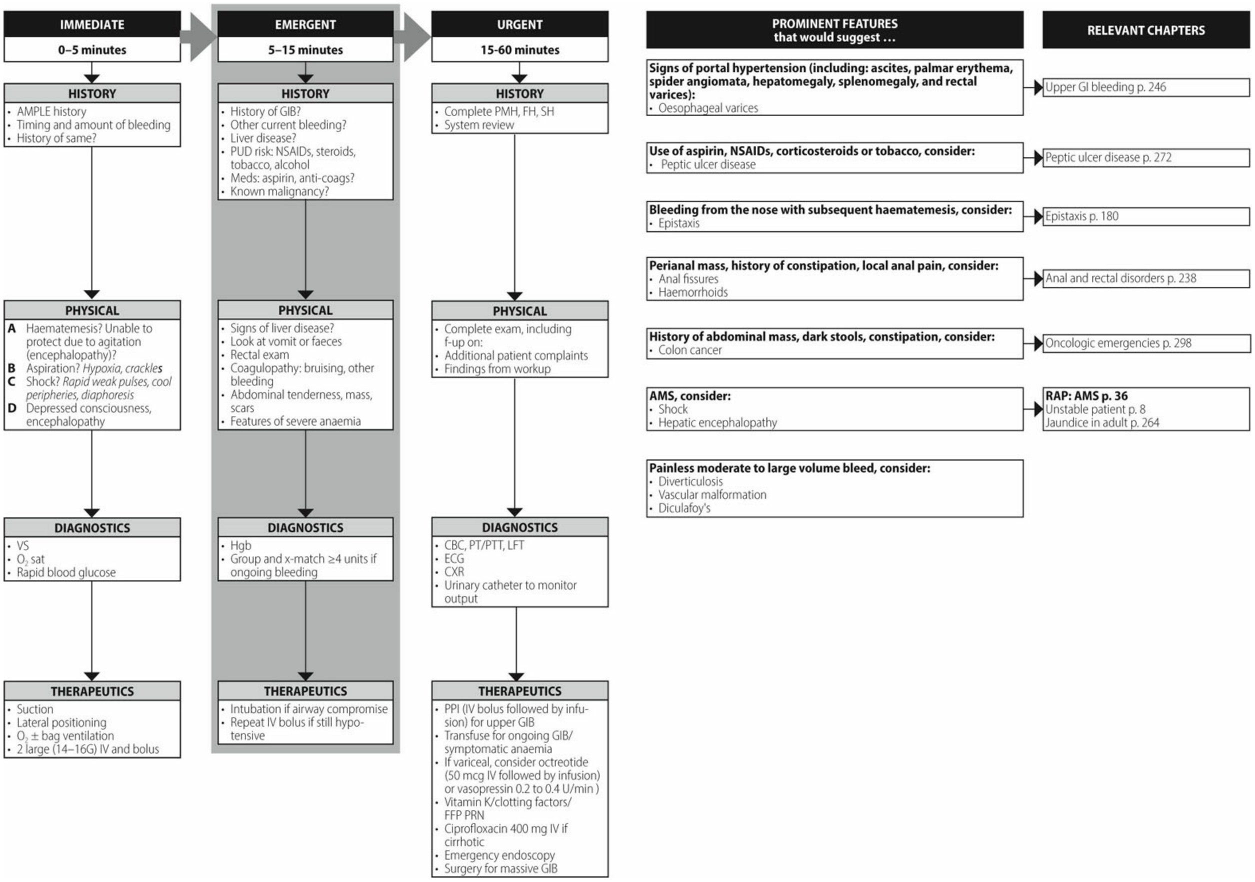
Follow-up on:
- Additional patient complaints
- Abnormal findings from Dx/W/Up

DIAGNOSTICS
- CBC, diff, smear, malaria slide
- Electrolytes, renal function, LFT, UA /culture
- Blood cultures
- LP with opening pressure (CT head prior if risk of ↑ICP)
- TSH, lactate, if available
- US: heart and IVC for volume status
- CXR (pneumonia?)
- CTH ± contrast if HIV and CNS involvement (fever, seizure, deficit)

THERAPEUTICS
- Repeat IV bolus if persistent hypotension
- Antibiotics and antimalarials empirically if critically ill, or per exam and testing
- Treat thyrotoxicosis per (📖 p. 410)

PROMINENT FEATURES that would suggest …	RELEVANT CHAPTERS
Persistent hypotension, tachycardia, shock, consider: • Septic shock, adrenal insufficiency, severe malaria, typhoid	Sepsis p. 318 Adult with fever p. 313 Fever in immunodeficiency p. 310 Adrenal insufficiency p. 414 Malaria p. 326 Typhoid p. 346
Bleeding, including petechiae, consider: • Dengue, meningococcaemia, DIC, TTP, toxic shock syndrome, viral haemorrhagic fever, Rickettsial disease	Dengue p. 374 Meningitis p. 330 DIC p. 288 Coagulopathies p. 288 TSS p. 380 VHF p. 372 Drug eruption p. 165 Tick-borne illness p. 376
Rash (non-purpuric), consider: • Dengue, drug eruption (TEN-SJS), Tick-borne illness, leptospirosis, chicken pox/varicella, HIV (maculopapular or Kaposi's)	Rash p. 144 Dengue p. 374 Tick-borne illness p. 376 Drug eruption p. 165
Possible tox, consider: • Anticholinergics, sympathomimetics (cocaine, amphetamine, PCP), serotonin syndrome, salicylates, petroleum distillates (gasoline, kerosene, ignitable liquids)	Toxic shock p. 380 Toxic traditional therapies p. 697 Toxic plants p. 719 Tox section chapters per specific agent pp. 652–724
Urinary findings: • Urinary tract infection (pyelonephritis may present with flank pain without dysuria), prostatitis, STI, PID	UTI in adults p. 604 Prostatitis p. 612 Pelvic pain p. 502 Vaginal discharge p. 500 STI p. 356
Respiratory symptoms: • Pneumonia, influenza, TB, pulmonary embolus, *Pneumocystis* pneumonia	Pneumonia p. 332 Influenza p. 340 TB p. 337 PE p. 118 HIV p. 320
Neurologic features, consider: • Meningitis (headache, neck stiffness), brain abscess, cerebral malaria (headache, AMS, or focal neuro), haemorrhagic stroke (headache, AMS, or focal neuro), subarachnoid haemorrhage (headache) • Spinal infections, Pott's (back pain, weakness), tetanus (tonic spasm), cavernous sinus thrombosis (headache, eye complaints)	Meningitis p. 330 Malaria p. 326 Spinal compressive lesions p. 460 SAH p. 468 Stroke p. 464 Spinal infections p. 364 Tetanus p. 362 Cerebral venous thrombosis p. 388
Back pain, consider: • Epidural abscess, diskitis, vertebral osteomyelitis, Pott's • Pyelonephritis • Psoas abscess • Dengue • Liver-gallbladder-pancreas pathology (may present as R flank pain)	UTI p. 604 Spinal infection p. 364 Dengue p. 374 Gallbladder p. 244 Pancreatitis p. 270
Abdominal pain, consider: • Typhoid (with or without diarrhoea) • Biliary disease (incl cholangitis) • Psoas abscess • Liver/colon: acute hepatitis, liver fluke, hepatosplenic abscess, diverticulitis, appendicitis	Abdominal pain p. 32 Appendicitis p. 234 Gallbladder p. 244 Typhoid and cholera p. 346 Hepatitis p. 344 Jaundice p. 264
Chest pain, CCF, or new murmur, consider: • Infectious endocarditis, myocarditis, pericarditis, pulmonary embolus	Endocarditis p. 100 Myocarditis p. 122 Pericarditis p. 124 PE p. 118
Cyclical fever, consider: • Malaria • Tick-borne illness • Dengue • Leptospirosis	Malaria p. 326 Tick-borne illness p. 376 Dengue p. 374
Weight loss, prolonged fever, LAN, consider: • TB, malignancy, fungal infection, osteomyelitis, endocarditis, typhoid, HIV	TB p. 337 Oncologic emergencies p. 298 Endocarditis p. 100 Typhoid and cholera p. 346 Fever in immunodeficiency p. 310
Fever with anaemia, consider: • Acute leukaemia/lymphoma, aplastic anaemia, malaria, sickle cell with infection, DIC, TTP	Oncologic emergencies p. 298 Malaria p. 326 Sickle cell p. 282 DIC p. 288 Coagulopathies p. 288
Sore throat, stridor, drooling/difficulty swallowing, change in voice, consider: • Pharyngitis, epiglottitis, peritonsillar abscess (visible on inspection), retropharyngeal abscess (oropharynx may appear normal)	ENT infections p. 170 Epiglottitis p. 176 Stridor p. 172

23 Gasrointestinal bleeding in adults



24 Headache

IMMEDIATE
0–5 minutes

HISTORY
- AMPLE history
- Head trauma?
- Fever?
- Seizures?

PHYSICAL
- **A** Emesis? Aspiration risk due to AMS?
- **B** Respiratory distress, hypoxia
- **C** Shock? *Hypotension, rapid weak pulses, cool peripheries, diaphoresis, tachycardia, pallor*
- **D** AMS/ALoC? Signs of trauma? Focal neuro deficits?

DIAGNOSTICS
- VS
- O_2 sat
- Rapid blood glucose

THERAPEUTICS
- Cervical spine stabilisation if trauma
- Suction
- O_2 ± bag ventilation
- Dextrose 50% 1 ampule or 10% (500 ml) if RBG ≤ 3.5

See also Non-traumatic headache, p. 450

EMERGENT
5–15 minutes

HISTORY
- Headache: location, onset, quality, severity, positional (worse lying down: ↑ICP). Similar prior events?
- Sudden? Time of onset to maximal pain seconds to minutes → SAH
- Visual or speech changes? Dizziness, weakness or numbness?
- Fever? HIV?
- Nausea/vomiting?
- Neck or back pain?
- Details of any trauma

PHYSICAL
Focused neurological exam:
- ↑ICP (Cushing's triad, unequal pupils, fundi), AMS, cranial nerves, motor, sensory, cerebellar, gait
- Nuchal rigidity
- Eye exam: redness, swelling, firm to touch, ↓vision, ↑IOP (acute glaucoma?)
- Trauma survey

DIAGNOSTICS
- HIV
- CT head if possible stroke or SAH

THERAPEUTICS
- Intubation for deteriorating AMS?
- Antibiotics if suspected infection
- Anti-pyretic
- IVF bolus 1 L if hypotensive
- ↑HOB 30° if ↑ICP
- Analgesia. Antiemetic for migraine (e.g. metoclopramide or prochlorperazine 10 mg IV)

URGENT
15-60 minutes

HISTORY
Detailed history, including:
- History of malignancy, TB, weight loss?
- Postpartum or currently pregnant?
- FH: migraine, aneurysm, renal failure, sudden death?
- SH: alcohol? Drugs? Trauma? Carbon monoxide exposure?
- Meds: antithrombotics?

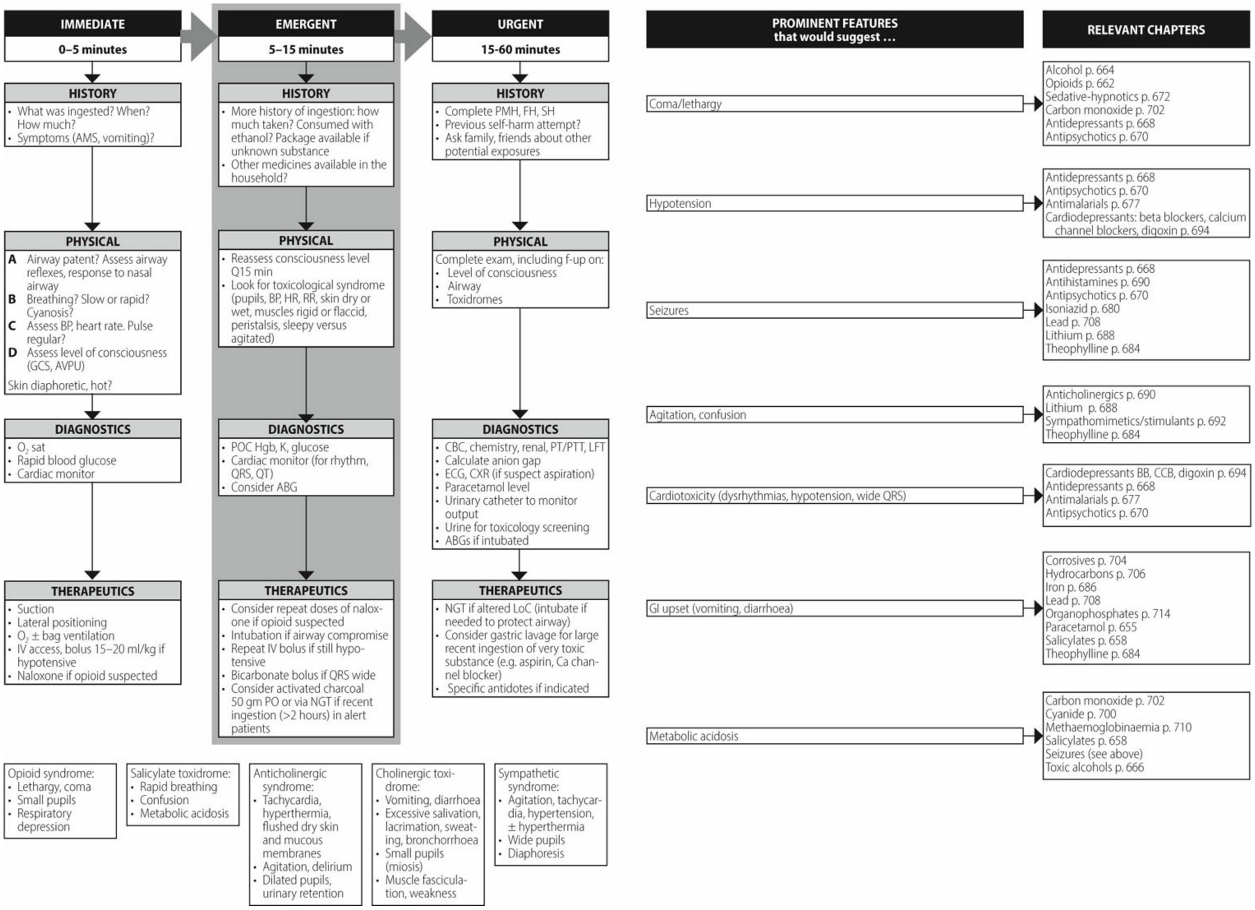
PHYSICAL
Complete exam, including:
- Secondary trauma survey, including otoscopy for haemotympanum
- Repeat neuro exam for evolving signs
- Skin for rash (infection, meningitis), red skin colour (late finding carbon monoxide)

DIAGNOSTICS
- CBC, electrolytes, renal function, PT/PTT, sed rate
- Malaria
- Urine pregnancy (eclampsia?)
- Blood cultures, urine culture if fever
- LP (meningitis, SAH)
- XR/CT c-spine if trauma
- CT/MRI brain for mass, oedema, stroke, malignancy, trauma

THERAPEUTICS
- Repeat IVF if hypotensive
- Antimalarials, antibiotics (+acyclovir if encephalitis or HIV)
- Opiate analgaesia if persistent pain
- Dexamethasone for tumour with↑ICP (call neurosurgery)
- Consider low-dose anti-HTN for BP≥180, decrease ≤ 20%

PROMINENT FEATURES that would suggest …	RELEVANT CHAPTERS
Sudden severe headache, consider: • SAH, carotid or vertebral artery dissection, cavernous (venous) sinus thrombosis, pituitary infarction, acute angle-closure glaucoma (red eye), hypertensive emergency	Subarachnoid haemorrhage p. 468 Stroke p. 464 Cerebral venous thrombosis p. 388 Adrenal and pituitary emergencies p. 414 Glaucoma p. 514 Hypertension p. 106
Fever or in HIV patient, consider: • Meningitis (incl cryptococcus), encephalitis, intra-cranial abscess, cerebral malaria, cavernous (venous) sinus thrombosis, toxoplasmosis	Meningitis p. 330 Malaria p. 326 Cerebral venous thrombosis p. 388
Visual changes, eye pain, tearing, consider: • Migraine, cluster headache, acute angle-closure glaucoma, temporal arteritis, SAH	Non-traumatic headache p. 450 Glaucoma p. 514 Vasculitis p. 646 Subarachnoid haemorrhage p. 468
Patient with HIV, cancer, or >50 years age, consider: • Intracranial mass lesion (infection, tumour)	Oncologic emergencies p. 298 HIV p. 320
Pregnant or post-partum, consider: • Eclampsia, pituitary apoplexy, cavernous (venous) sinus thrombosis, post-dural puncture, tension	Hypertensive disorders during pregnancy p. 482 Adrenal and pituitary emergencies p. 414 Cerebral venous thrombosis p. 388
AMS or focal neurologic signs/symptoms, consider: • Meningitis, encephalitis, cerebral malaria, brain mass/abscess • Stroke, SAH, ICH, atypical migraine • Carbon monoxide or other tox, vertebral/carotid dissection, hypertensive emergency • Cavernous sinus thrombosis, hypoglycaemia	Meningitis p. 330 Malaria p. 326 Carbon monoxide p. 702 Toxic shock p. 380 Subarachnoid haemorrhage p. 468 Altitude sickness p. 200 Cerebral venous thrombosis p. 388

25 Toxic ingestion



26 Ischaemic limb

IMMEDIATE
0–5 minutes

HISTORY
- AMPLE history
- Onset, progression, duration
- Abdominal pain? (aortic dissection)
- Trauma?

PHYSICAL
- **A** Airway clear and protected?
- **B** Hypoxia, DIB?
- **C** Shock? Pulses symmetrical?
- **D** AMS , motor/sensation of affected limb

DIAGNOSTICS
- VS
- O_2 sat
- RBG if AMS

THERAPEUTICS
- Suction
- Lateral positioning
- O_2 ± bag ventilation

EMERGENT
5–15 minutes

HISTORY
- Pain: acute or chronic, location, severity, aggravating factors (exertion?), mitigating factors (position of comfort)
- Diabetes, known coronary disease, palpitations, history of stroke
- Weakness or numbness in extremity
- Fever (endocarditis/septic emboli)
- Trauma

PHYSICAL
- Compare limbs
- Acute ischaemia: red/purple or pale, cool, painful, absent pulses, decreased power
- Chronic ischaemia: dusky erythema or black, cool, absent pulses, less painful, chronic wounds, bilateral changes
- Wet gangrene: blisters, oozing, erythema surrounding darkened skin
- Look: colour? Fracture or dislocation?
- Feel: temperature? Tender? Crepitus?
- Palpate pulses
- Test motor and sensory
- Auscultate heart for rhythm, murmur (dissection, emboli)
- Palpate for pulsatile mass (AAA)

DIAGNOSTICS
- Bedside doppler if pulses not palpable
- Bedside US: AAA?
- Blood for crossmatch (if trauma or suspected aortic aetiology)

THERAPEUTICS
- Analgaesia (morphine 4 mg IV/IM)
- Reduce and splint fracture or dislocation

URGENT
15-60 minutes

HISTORY
- Complete history
- Risk factors: diabetes, smoking, HTN, high cholesterol, atrial fib, stroke, AAA, alcohol use
- Poorly healing wounds
- Back pain

PHYSICAL

Complete exam including:
- Signs of chronic venous insufficiency: distal hair loss, trophic skin changes, hypertrophic nails, chronic wounds
- Other embolic phenomena: digital ischaemia or necrosis, splinter haemorrhages

DIAGNOSTICS
- CBC, blood grouping (if trauma or suspected aortic injury), electrolytes, renal function, PT/PT/PTT
- 12-lead ECG (atrial fibrillation?)
- CXR (aortic dissection?)
- Duplex US
- CT angiography
- Arteriography

THERAPEUTICS
- Antibiotics if suspected infection
- For thromboembolic ischaemia: heparin 80 u/kg IV bolus, then 18 u/kg IV infusion, adjust dosing per nomogram
- Urgent surgical consultation for:
  - » Compartment syndrome (fasciotomy)
  - » Any asymmetric ↓pulses or new sensory changes associated with pain
  - » Acute (<2 weeks) limb ischaemia or wet gangrene
  - » AAA or aortic dissection

PROMINENT FEATURES that would suggest … / RELEVANT CHAPTERS

PROMINENT FEATURES that would suggest …	RELEVANT CHAPTERS
Abdominal pain, abdominal mass, flank pain, or back pain, consider: • Abdominal aortic aneurysm ⟶ peripheral vascular compromise	Abdominal aortic aneurysm p. 112
Limb trauma with reduced pulses, consider: • Fracture or dislocation with arterial injury • Fracture or dislocation with venous injury • Compartment syndrome	ATLS p. 726 Peripheral vascular trauma p. 770 Compartment syndrome p. 566
Chest pain, consider: • Aortic dissection with flap causing vascular compromise	Aortic dissection p. 114
Fever, consider: • Wet gangrene • Necrotising fasciitis • Endocarditis with septic emboli	Approach to sepsis p. 318 Cellulitis p. 378 Infectious endocarditis p. 100
Bilateral lower limb ischaemia, consider: • Chronic vascular insufficiency (diabetes, peripheral vascular disease) • AAA or other obstruction proximal to iliac bifurcation • Hypercoagulable states	Abdominal aortic aneurysm p. 112

27 Seizure in children

IMMEDIATE
0–5 minutes

HISTORY
- AMPLE history: seizures?
- Onset, progression, duration
- Responsive during event?
- Description of movements, bilateral?

PHYSICAL
- **A** Emesis? Aspiration risk?
- **B** Aspiration? Hypoxia, crepitations
- **C** Shock? *Hypotension, rapid weak pulses, cool peripheries, diaphoresis, tachycardia, pallor*
- **D** AVPU
- Ongoing seizure? *AMS, deviated eyes, rhythmic movements, sudden tachycardia*
- Increased ICP? *Hypertension with bradycardia (Cushing's), unequal pupils, posturing*
- Focal deficit?

DIAGNOSTICS
- Rapid blood glucose
- VS
- O_2 sat

THERAPEUTICS
- Suction
- C-spine immobilisation if trauma
- Lateral positioning
- O_2 ± bag ventilation
- Dextrose 10% (5 ml/kg) if RBG≤3.5
- No IV/IO access: 5 ml/kg 10% dextrose via NGT, or 1 ml of 50% dextrose in buccal space (between lower gum and cheek) Q3–5 minutes

Benzodiazepines
- IV
  - » Lorazepam (0.1 mg/kg)
  - » Midazolam (0.2 mg/kg)
  - » Diazepam (0.3 mg/kg)
- NO IV
  - » Lorazepam (0.1 mg/kg) buccal
  - » Midazolam (0.5 mg/kg) buccal
  - » Diazepam (0.5 mg/kg) rectal

EMERGENT
5–15 minutes

HISTORY
- Fever, illness, PO, UOP?
- Ingestions (toxins)
- Patient or household contact on TB therapy (INH)?
- Occult trauma
- Preceeding events
- On seizure meds? Missed dose?

PHYSICAL
- Monitor ABC after meds
- Ongoing seizure (subclinical)?
- Focal neuro findings?
- Evidence of infection? Rash?
- Evidence of trauma?

DIAGNOSTICS
- Recheck glucose
- Recheck O_2 sat and BP

THERAPEUTICS
- Repeat benzo dose
- Intubation or bag ventilation for hypoventilation
- Still seizing 10 min after 2nd benzo dose:
  - » Phenobarbitone (monitor BP/RR): 20 mg/kg IV slow bolus or IM. If no IV form, give crushed oral tabs via NGT OR
  - » Phenytoin 20 mg/kg IV over 20 min, cardiac monitor
- If possible INH toxicity, treat with pyridoxine (B_6): 70 mg/kg IV or 1 gm per gram of INH ingested if known

URGENT
15–60 minutes

HISTORY
- Complete history of any recent illness and treatment
- Complete PMH including birth history
- SH: any toxins or meds in home?
- FH: epilepsy or febrile seizures?

PHYSICAL
- Complete physical exam including skin
- Full CNS exam as possible for mental status
- Head circumference, weight
- Dysmorphology

DIAGNOSTICS
- LP: measure CSF pressure, send CSF studies; consider HSV PCR
- Malaria (RDT/smear), CBC, electrolytes, blood culture, seizure med levels, tox screen
- CT if focal sx, persistent ↓LoC
- EEG if concerned sub-clinical seizure

THERAPEUTICS
Still seizing:
- If phenobarb loaded, give additional phenobarb dose (10 mg/kg IV/IM/NG). CAREFUL BP/RR MONITORING
- If phenytoin loaded, start midazolam infusion (0.001 mg/kg/min, increase every 15 min by 0.001 mg/kg/min until either seizures stop or max dose 0.008 mg/kg/min)
- Prepare for intubation/ICU transfer
- Correct electrolytes
- IV antibiotics if indicated

PROMINENT FEATURES that would suggest … → RELEVANT CHAPTERS

Fever, consider:
- CNS infection: encephalitis, meningitis, cerebral malaria
- Simple febrile seizure: only if well-appearing, aged >6 months and <6 years, seizure under 15 min with NO focal features, and no prior afebrile seizures

RAP: Fever p. 62
Crying or irritable child p. 48
Approach to the child with fever p. 316
Malaria p. 326
Meningitis p. 330
TB p. 337
Lumbar puncture p. 826

History of epilepsy, consider:
- Factors lowering seizure threshold (infection, insomnia, puberty)
- Missed dose of seizure meds
- Progressive disease, need for new medication regimen
- Undiagnosed space-occupying lesion if no previous MRI
- Undiagnosed metabolic or thyroid disease

Paediatric oncologic presentations p. 304
Thyroid disorders p. 410, p. 412
Adrenal and pituitary emergencies p. 414
Reading the head CT p. 954

Refractory seizures, consider:
- Electrolyte disturbance: hypernatraemia, hyponatraemia
- Venous sinus thrombosis
- Toxin exposure (especially INH)

Sodium disorders p. 418
INH toxicity p. 680
Toxic shock p. 380
Adrenal and pituitary emergencies p. 414
TB meds p. 680
Toxic effects of traditional therapies p. 697

Inconsistent history, other injuries, consider:
- Non-accidental CNS trauma

Approach to suspected child abuse p. 872
Approach to paediatric trauma p. 730
Head injury p. 755

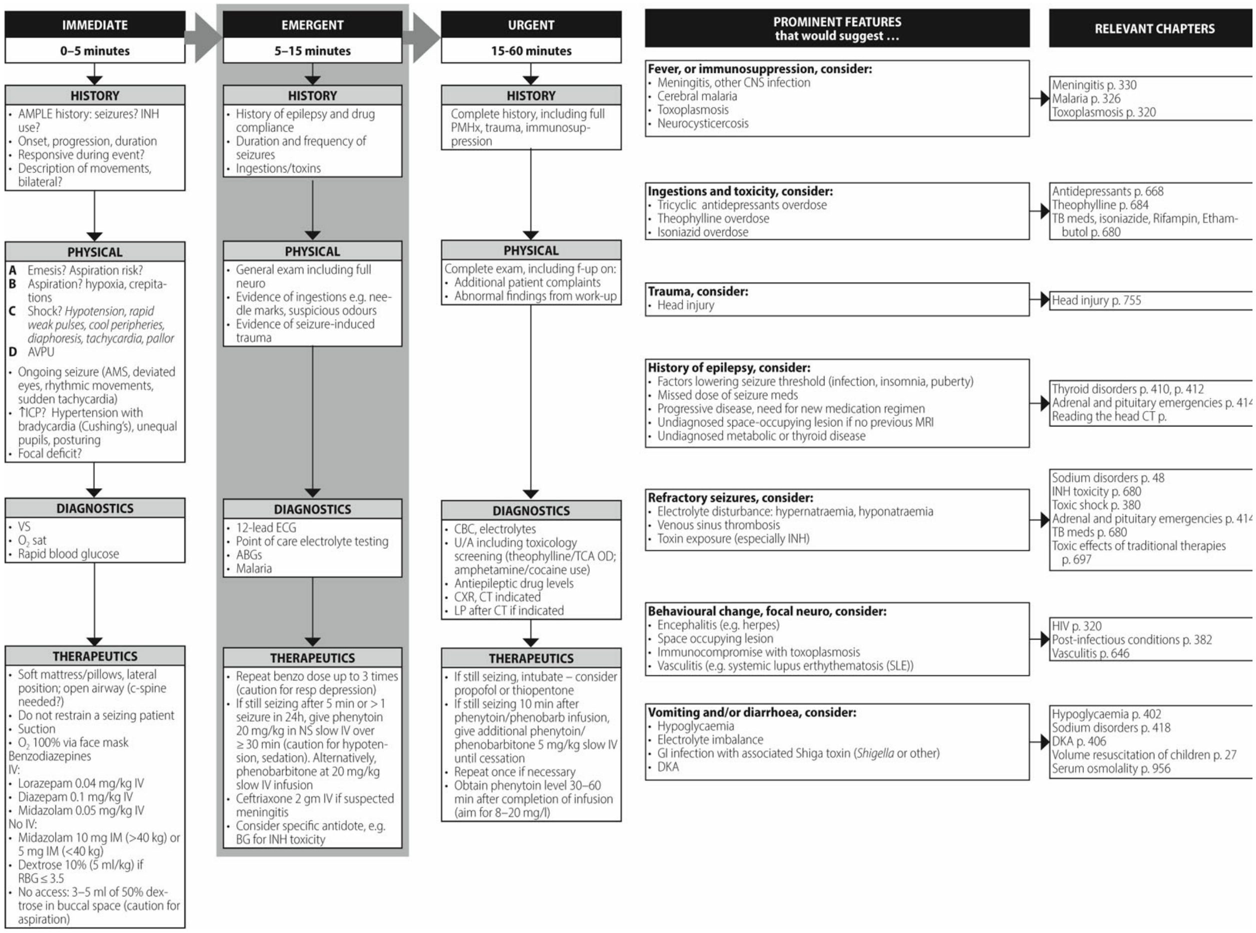
Behavioural change, focal neuro, consider:
- Encephalitis (e.g. herpes)
- Space occupying lesion
- Immunocompromise with toxoplasmosis
- Vasculitis (e.g. systemic lupus erthythematosis (SLE))

Pediatric oncologic presentations p. 304
HIV p. 320
Post-infectious conditions p. 382
Vasculitis p. 646

Vomiting and/or diarrhoea, consider:
- Hypoglycaemia
- Electrolyte imbalance
- GI infection with associated Shiga toxin (*Shigella* or other)
- DKA

Hypoglycaemia p. 402
Sodium disorders p. 418
DKA p. 406
Volume resuscitation of children p. 27
Serum osmolality p. 956

28 Seizure in adults



29 Syncope

IMMEDIATE — 0–5 minutes

HISTORY

- AMPLE history
- Chest or abdominal pain or DIB?
- Seizure activity?
- Possibly pregnant?
- GI bleed/black stools?

PHYSICAL

- **A** Aspiration risk (due to AMS, unresponsive)?
- **B** Hypoxia, wheezing, distress
- **C** Shock? *Hypotension, rapid weak pulses, cool peripheries, diaphoresis, tachycardia, pallor*
- **D** AMS? Obvious injury from syncopal collapse?

DIAGNOSTICS

- VS
- O_2 sat
- Rapid blood glucose

THERAPEUTICS

- Secure c-spine if history trauma or fall with syncopal event
- Suction
- Lateral positioning
- O_2 ± bag ventilation
- Dextrose 50% 1 ampoule (adult), dextrose 10% 5 ml/kg (paediatric) if RBG ≤ 3.5

EMERGENT — 5–15 minutes

HISTORY

- Activity and symptoms immediately preceding, duration of LoC, witnessed seizure activity, injury, prodrome (nausea, sweating, dizziness, visual or hearing changes)?
- History of syncope
- Associated palpitations
- Headache? Fever? Diabetic medication?
- Abdo or back pain (aortic path)?
- Focal weakness or numbness, focal neurologic symptoms?
- Ingestion, toxins

PHYSICAL

- AMS
- Cardiac: JVP, murmur, normal pulse?
- Pulm: ↑work of breathing, creps, wheezes
- Abdomen: tenderness, distension, rebound, pulsatile mass
- Neuro: mental status, facial asymmetry, extremity weakness (stroke? SAH?)
- Extremities: obvious injury or deformity due to syncopal fall? Unilateral calf pain or swelling (DVT/PE)?
- Rectal for food

DIAGNOSTICS

- 12 lead ECG
- Crossmatch blood if signs of haemorrhage
- Urine pregnancy in haemodynamically unstable female patient

THERAPEUTICS

- Intubation if airway compromise
- IV bolus 1 L if hypotension
- Trendelenberg positioning if hypotensive but protecting airway

URGENT — 15-60 minutes

HISTORY

Complete history:
- Emotional stressors
- Eating or drinking less than usual
- Concurrent illness
- Change in medications
- Drug or alcohol use
- History cardiac disease, CCF
- Bleeding from nose, mouth, emesis, rectum, vagina
- Family history of sudden death

PHYSICAL

- Complete exam and trauma eval (injuries from syncopal collapse?)

Follow-up on:
- Additional patient complaints
- Abnormal findings from work-up

DIAGNOSTICS

- Repeat glucose
- CBC, electrolytes, renal function
- Cardiac enzymes, D-dimer if chest pain or dysnpoea
- UA and urine pregnancy
- CXR, XR/CT c-spine if trauma
- US for hypotension (AAA, CCF, PTX)
- LP (SAH)
- CT head for AMS, head trauma, severe headache (ICH)
- CT chest and/or US if PE suspected
- Echocardiogram

THERAPEUTICS

- Repeat IV bolus if persistently hypotensive
- Dextrose 50% 1 ampoule (adult) or 10% 500 ml IV if RBG < 3.5
- Transfuse as indicated
- Treat arrhythmias

PROMINENT FEATURES that would suggest … / RELEVANT CHAPTERS

PROMINENT FEATURES that would suggest …	RELEVANT CHAPTERS
Abdominal pain or tenderness, consider: • AAA, GI bleeding • Ectopic pregnancy, sepsis with intra-abdominal source	Abdominal aortic aneurysm p. 112 Upper GI bleeding p. 246 Lower GI bleeding (including diverticulitis) p. 248 Ectopic pregnancy p. 480 Bowel obstruction p. 236 Approach to abdominal pain p. 32
Headache, focal neuro signs, or AMS, consider: • SAH, stroke, SOL, head injury • Meningitis/encephalitis, malaria • Toxic ingestion	**RAP: AMS p. 36** Stroke p. 464 Transient ischaemic attack p. 110 Subarachnoid haemorrhage p. 468 Malaria p. 326 Meningitis p. 330 Toxic shock p. 380
Chest pain, DIB, palpitations, oedema, consider: • MI, DVT/PE, constrictive pericarditis, tamponade, aortic dissection • Critical AS, IHSS/HOCM, pulmonary hypertension, cardiomyopathy, CCF	DVT/PE, tamponade, dissection, cardiomyopathy, CCF pp. 84–127
Persistent hypotension, consider: • Shock (septic, cardiogenic, neurogenic, hypovolaemic) • Severe dehydration • Anaemia • GIB, trauma	Sepsis p. 318 Cardiogenic shock p. 88 Adult resus p. 8 Anaemia p. 285 GIB p. 246, p. 248 ATLS p. 726
Stridor, wheezing, or respiratory distress, consider: • Choking • Anaphylaxis • Upper airway obstruction • Status asthmaticus	**RAP Anaphylaxis p. 38** Choking p. 10 Stridor p. 172 Asthma p. 626
Electrolyte abnormalities: • Hypoglycaemia • Hyperthyroidism • Adrenal insufficiency • Dehydration • Malnutrition • Vomiting • Diarrhoeal illness	Hypoglycaemia p. 402 Hyperthyroidism p. 410 Adrenal insufficiency p. 414 Vomiting p. 274 Diarrhoea p. 242
Headache or neck pain, consider: • C-spine injury, blunt head injury, carotid dissection, SAH	Approach to neuro-imaging p. 734 Head injury p. 755
Tongue wound, urinary incontinence, prolonged confusion, consider: • Seizure • Toxic ingestion	**RAP: Seizures p. 76** Toxic shock p. 380
Prodrome of dizziness, nausea, visual blurring; very hot or dehydrated; emotional stressors; consider: • Vasovagal syncope • Heat-associated illness	Syncope p. 454 Heat-associated illness p. 202

30 Weakness in children

IMMEDIATE
0–5 minutes

HISTORY
- AMPLE history
- Onset, duration?
- Trauma? Seizure?
- Difficulty breathing
- Fever

PHYSICAL
A Pooling secretions?
B Hypoxia, rapid or shallow breathing, respiratory distress?
C Shock? *Low BP, rapid weak pulses, cool peripheries, diaphoresis, tachycardia, pallor*
D Severe weakness: unable to lift head, diaphragmatic breathing, inability to suck, agitation, anxiety

DIAGNOSTICS
- O_2 Sat
- VS
- RBG

THERAPEUTICS
- C-spine immobilisation if trauma
- Airway repositioning and NPO until swallow study
- O_2 ± bag ventilation
- IV and bolus 20 ml/kg if shocked
- Dextrose 10% (5 ml/kg) if RBG ≤ 3.5

EMERGENT
5–15 minutes

HISTORY
- Onset, duration, pattern (ascending or descending?), speed (rapid or gradual?). Previous episodes?
- Back/neck/muscle pain?
- Tearing, drooling, diarrhoea, secretions (organophosphates)?
- Preceding events/symptoms:
  - » Ingestion: contaminated food, fish, shellfish. Honey or canned foods (botulism?)
  - » Animal or insect bites/stings
  - » Fever or recent illness? URI? Vomiting/diarrhoea? Recent immunisation?
  - » Recent back or spine procedures?

PHYSICAL
- Mental status
- Respiratory: muscle weakness?
- Neuro:
  - » Eyes: pupils, extraocular movements
  - » Cranial nerves
  - » Motor (strength, tone, reflexes, symmetry)
  - » Sensation loss (distinct level?)
  - » Cerebellar
  - » Gait
- GU: urinary retention, rectal tone
- Musculoskeletal: midline spinal tenderness or deformity. Muscular tenderness? Fasciculations?

DIAGNOSTICS
- ABG/VBG if respiratory symptoms
- Serum K, Mg, Ca
- ECG: rhythm, intervals (electrolytes? toxins?)

THERAPEUTICS
- Consider intubation if weak resp effort
- If Organophosphates: avoid suxamenthonium and give atropine 0.01mg/kg IV 📖 p. 714
- Consider Foley catheter

URGENT
15–60 minutes

HISTORY
Full history, including:
- PMH: developmental milestones, asthma, Down's syndrome, hypothyroid, rickets
- Meds: steroids?
- If recurrent: triggers (exercise, fasting?)
- Immunisation status (polio, diphtheria)
- HIV or other immunocompromise?

PHYSICAL
Full exam, including:
- FULL skin exam. Pallor? Rash or eschar? Wound? Look for and remove ticks
- Evidence of longstanding weakness: atrophy, contractures
- Dysmorphology; small for age
- Nutritional deficits (rickets, bruising)

DIAGNOSTICS
- CBC, electrolytes, renal function, CK, UA, stool culture
- Respiratory function testing (e.g. NIF)
- CXR, spine XR
- Lumbar puncture
- CT brain (space occupying lesion, stroke)
- CT or MRI spine (ADEM, cord compression)
- Nerve condution studies (GBS)

THERAPEUTICS
- Replete electrolytes as needed
- Analgesia
- Supportive: respiratory (NIPPV, intubation), IVF and vasopressors for BP, urine catheter
- Early Neurology and Neurosurgery involvement
- Specific therapies:
  - » Antibiotics
  - » Botulinum antitoxin
  - » Intravenous immunoglobulin (GBS, botulism)
  - » Anti-venom (snake bite)
  - » Brain protective measures (stroke, trauma): normothermia, normocarbia, treat seizures, elevate head of bed 30°, monitor for ↑ICP

PROMINENT FEATURES that would suggest … → RELEVANT CHAPTERS

History of trauma:
- Sudden onset, normal mental status, consider:
  - » Spinal cord injury: compression fracture or dislocation, cord concussion/infarction, SCIWORA
- Gradual onset or associated AMS, consider:
  - » Intracranial causes: ICH, SDH

→ **RAP: AMS p. 34**
Spinal injury p. 768
Spinal compressive lesions p. 460
Head injury p. 755

Non-traumatic flaccid paraparesis with sensory level, ± bladder/bowel, consider:
- Acute transverse myelitis
- Compressive myelopathy: spinal TB, trauma, tumour, abscess, haematoma
- Acute demyelinating encephalomyelitis (ADEM)
- Infectious myelitis: HIV, herpes zoster myelitis, TB radiculomyelitis

→ **RAP: AMS p. 34**
Transverse myelitis p. 456
Spinal compressive lesions p. 460
Spinal injury p. 768
TB p. 337
Post infectious conditions p. 382
Varicella p. 342
HIV p. 320

Flaccid, afebrile symmetric para/quadriparesis (+/- bulbar and respiratory weakness), but no sensory level, consider:
- Guillain Barré
- Toxins/poisoning (ingestions, tick paralysis, envenomation, marine stings)
- Diphtheria
- Myasthenia gravis
- Botulism

→ Botulism p. 360
Approach to weakness p. 432
Peripheral nerve disorders p. 445
Tick borne illness p. 376
Organophosphates p. 714
Post infectious conditions p. 382
Marine stings p. 216
Spider bites p. 226
Snake bites p. 224

Fever and acute asymmetric flaccid paralysis, no sensory level or bladder involvement, consider:
- Enteroviral myelitis: coxsackie virus, echovirus
- Poliomyelitis or vaccine-associated poliomyelitis

→ Approach to weakness p. 432
Spinal cord infections p. 364

Recurrent episodes of weakness, consider:
- Metabolic neuropathies: acute intermittent porphyria (associated abdo+/-limb pain), hereditary tyrosinemia (associated pain, axial hypertonicity)
- Periodic paralyses: familial hypokalaemic periodic paralysis
- Familial hyperkalaemic periodic paralysis
- Demyelinating conditions: multiple sclerosis

→ Approach to weakness p. 432
Peripheral nerve disorders p. 445
Potassium disorders p. 426

Proximal muscle weakness, muscle tenderness, preserved reflexes, no sensory signs or symptoms, consider:
- Myositis (acute infectious): viral, pyomyositis
- Rhabdomyolysis: after prolonged seizures, envenomation, trauma. Urine dip (+) blood, no RBC on microscopy, CK elevated, acute kidney injury

→ Approach to weakness p. 432

Focal weakness only (hemiplegia) consider:
- Stroke: ischaemic, haemorrhagic
- Stroke mimics: tumour, trauma, hypoglycaemia, hemiplegic migraine

Focal weakness only (monoplegia), consider:
- Asthmatic amyotrophy: acute, preceding asthma within 10 days, neurotropic viruses
- Acute idiopathic plexitis (brachial or lumbar): acute, immune-mediated, severe pain, ± fever
- Poliomyelitis

Generalised, non-specific weakness or acutely floppy child, consider:
- Electrolytes: hypokalaemia, hypocalcaemia, severe dehydration
- Sepsis: particularly in malnourished child
- Flaccid encephalopathy from any acute severe brain illness or injury (eg, cerebral malaria); congenital metabolic disorders

→ **RAP: AMS p. 34**
RAP: Dehydration p. 50
Stroke p. 464
Potassium disorders p. 426
Calcium disorders p. 422
Approach to SAM p. 394
Diarrhoea p. 242

Inability to walk/stand but normal neuro, consider:
- Septic arthritis
- TB osteitis
- Diskitis: uncommon, more likely if immunocompromised and <3years old, *Staph aureus* most common (pain prominent, especially with spinal flexion, ± fever)

→ Spinal cord infections p. 364
Arthritis p. 640
TB p. 337

Part

4

A. Cardiovascular system

31 ECG basics and vascular localisation

The electrocardiogram (ECG) is a diagnostic tool that measures and records the electrical activity of the heart muscle as it changes with time. ECG interpretation provides a valuable diagnostic aid in life-threatening cardiac conditions.

Obtaining the ECG

Standard ECG consists of 12 leads – six limb leads and six precordial leads.

Lead placement

Limb leads

Leads I, II, III, aVR, aVL, aVF represent electric potentials between limb leads

- R Arm/aVR: upper right arm, away from thick muscle

• L Arm/aVL: upper left arm, away from thick muscle
• R Leg/N: inner calf of right leg
• L Leg/aVF: inner calf left leg

Precordial leads

V1: fourth intercostal space to the right of the sternum
V2: fourth intercostal space to the left of the sternum
V3: midway between leads V2 and V4
V4: fifth intercostal space at mid-clavicular line
V5: midway between V4 and V6
V6: fifth intercostal space in mid-axillary line

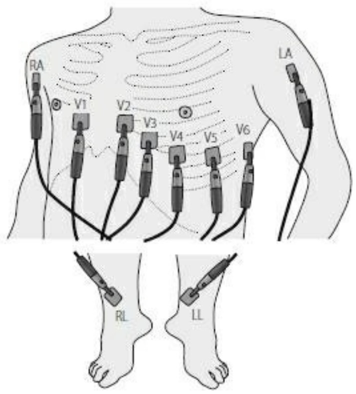


Figure 31.1 ECG Lead placement on a patient

ECG recording

• A plot of voltage on the vertical axis against time on the horizontal axis
• ECG paper speed is 25 mm/sec
• Small horizontal box (light lines) of 1 × 1 mm corresponds to 0.04 seconds
• Large horizontal box (heavy lines) of 5 × 5 mm corresponds to 0.2 seconds
• Small vertical square of 1 × 1 mm represents 0.1 mV
• Large vertical square of 5 × 5 mm represents 0.5 mV

Electrical activity:
• P-wave: atrial depolarisation
• PR interval: AV conduction system
• QRS: ventricular depolarisation
• T: ventricular repolarisation

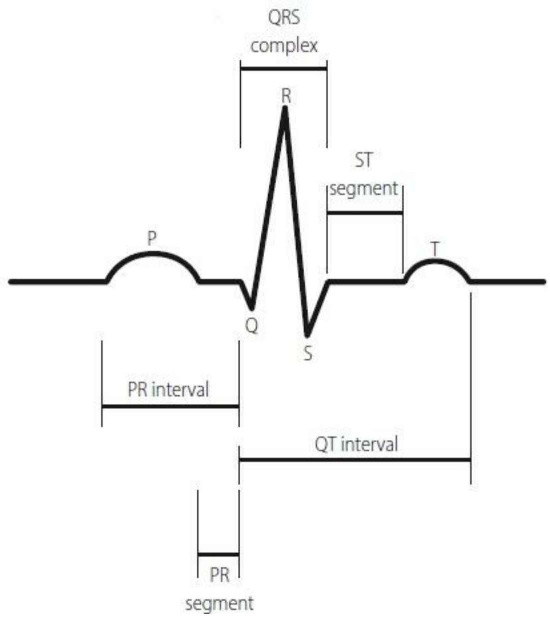


Figure 31.2 ECG wave forms and intervals

ECG interpretation

Check to confirm the patient's name, age and the ECG date.

Rate: *Fast, normal, or slow*?

- Normal (60–100)
- Bradycardia (< 60), tachycardia (> 100)
  - » Manual calculation of rate:
    - Length of the average ECG: 30 big squares = 6 sec
      Count the number of beats and multiply by 10.
    - Atrial rate = (number of P-waves 6-second strip) × 10
    - Ventricular rate = (number of R-waves 6-second strip) × 10

Rhythm

- Regular or irregular?
  - » In a regular rhythm, the interval between R waves will be equal
- P-WAVE: *is the rhythm sinus or not sinus*?
  - » If **sinus**, a P-wave should precede each QRS and a QRS follow each P
  - » Intermittent dissociation between P and QRS (some P-waves transmitted with QRS following, some not) suggests **second-degree heart block**
  - » Complete dissociation between P-waves and QRS complexes suggests **third-degree heart block**
  - » Absent P-waves with irregularly irregular QRS complex suggests **atrial fibrillation**
  - » Continuous, regular, saw-toothed pattern P-waves at rate 250–350 with regular QRS intervals at rate divisive of 300 (rate 300, 150, 75) suggests **atrial flutter**
- QRS complex: *is the QRS complex wide or narrow?*
  - » Normal QRS is less than 0.12s (three small boxes)
  - » Wide QRS indicates ventricular pacemaker or conduction defects
    - Left or right bundle branch block
    - Hyperkalaemia
    - Toxic ingestion
    - **Ventricular tachycardia**, if wide and fast

Axis

- Is there a right or left axis deviation? Which way does the QRS point?
- QRS up (positive) in leads I and AVF: normal axis
- QRS up in I and down in AVF: left axis deviation. Likely due to left anterior fascicular block
- QRS down in I and up in AVF: right axis deviation. Likely due to right ventricular hypertrophy

Intervals (see Figure 31.2)

- PR interval: *how long is the PR interval?*
  - Constant PR is 0.12–0.2s
  - Short PR indicates fast AV conduction through accessory pathway (e.g. Wolff-Parkinson-White syndrome)
  - Long PR interval with P for every QRS complex and QRS for every P-wave indicates first degree heart block
  - Progressive prolongation of PR interval with subsequent dropping of QRS is Mobitz type I second-degree block
  - Constant PR interval with intermittent dropping of QRS is Mobitz type II second-degree block
- QT interval: *prolonged?*
  - Many ECG readings report QT corrected for rate (the QTc)
  - May be calculated online at ecgpedia.org
  - Hodge formula for QTc = QT + 1.75 (HR–60)
  - QTc > 500 ms is prolonged
  - QTc prolongation due to: medications (antihistamines, macrolides, phenothiazine, calcium channel blockers, quinidine), acute myocardial ischaemia, myocarditis or electrolyte imbalance (low K, Ca or Mg)

Ischaemia

- Q waves: *are there Q waves?*
  - Normal Q waves are less than 0.04 s (one small box) long and less than 0.2 mV deep
  - Pathological Q waves may appear after acute infarction
- ST segment: *is the ST segment elevated or depressed?*
  - Isoelectric baseline defined by segment between the end of the T-wave and the following P
  - Normal ST is isoelectic
  - **ST elevation** > **1 mm** suggests acute infarction
  - **ST depression > 0.5 mm** suggests subendocardial ischaemia or reciprocal changes
  - ST elevation, especially if in **all or most** leads, may represent nonischaemic cardiac conditions, including bundle branch block, LVH, pericarditis, and pacemaker
  - Consider the clinical presentation – chest pain, dyspnoea, hypotension, distress – when interpreting the meaning of ST elevation
- T-waves: *what is the morphology of T-wave?*
  - Normal T-wave is asymmetric, smooth with variable amplitude
  - T-wave peaking (hyperacute T-waves) suggests acute myocardial ischaemia and presents before ST elevations
  - Diffuse T-wave peaking suggests severe hyperkalaemia
  - T-wave flattening suggests hypokalaemia or digitalis toxicity

Cardiac vascular localisation

ECG can suggest coronary artery occlusions. ST elevation in **two contiguous leads** localises the lesion to an anatomic segment of the heart.

Area of ST segment elevation	**Leads defining this area**	**Culprit artery**
Anterior	V1–V4	98% LAD
Apical	V5–V6	25% LAD 62% RCA 13% LCX
Lateral	I, aVL, V5–V6	63% LAD 37% LCX

Inferior	II, aVF, III	85% RCA 14% LCXR
Posterior	ST depression V1–3	Usually LCX

RCA = right coronary artery; LCX = left circumflex artery; LAD = left anterior descending artery

32 Approach to the patient with cardiogenic shock

Shock (inadequate tissue perfusion caused by circulatory insufficiency) has many causes. When the primary cause is cardiac dysfunction, the shock is cardiogenic. This may be due to poor contraction (ischaemia, myocarditis), abnormalities of rate and rhythm (tachy- and brady-arrhytmias), obstruction to outflow (stenotic valvular lesions), or other valvular abnormalities (acute valvular incompetence). Cardiac tamponade is sometimes classified as a form of cardiogenic shock, but is treated separately here (📖 p. 102). Finding and reversing the specific cause is essential as poorly treated cardiogenic shock has a high mortality rate.

The first five minutes

- ABC, VS, O_2, IVF, cardiac monitor
- ECG
- Bedside US

History and physical examination

Key historical features

These patients are acutely and severely unwell – a full history may not be possible.

Onset and duration of symptoms, pre-existing diseases, medication use, cardiac risk factors, family history.

Signs and symptoms

- Patient may present with dyspnoea, AMS, chest pain, palpitations, angor animi (sense of impending death), and collapse
- VS: compensatory tachycardia (although bradycardia may be primary cause), hypotension. Pallor and diaphoresis. Weak pulses. Features of cardiac failure (pulmonary crackles, raised JVP, S3 gallop, peripheral oedema)
- Search for features of a possible cause: fever or infectious symptoms → myocarditis; conjunctival pallor and splenomegaly → anaemia; muffled heart sounds and JVD → tamponade

Differential diagnosis and possible causes

Consider all other causes of shock states (i.e. hypovolaemia, anaphylaxis, sepsis, toxidromes, envenomations, acute severe hypertension) and cardiac failure.

Investigations

Bedside US, ECG and CXR provide the most useful initial information. Consider further testing based on the suspected cause.

- Labs: CBC, electrolytes, renal, ABG ◇; cardiac enzymes ◈
- ECG ◇: (ischaemia, dysrhythmias, ventricular hypertrophy and strain)
- Imaging: CXR ◇ (pulmonary congestion, pleural effusion, cardiomegaly, pneumonia, PTX); bedside US ◈ (dyskinesis, LV or RV dilatation, valvular insufficiency, mitral regurgitation or tricuspid insufficiency, pericardial effusion)

Treatment

The goal of acute management is to restore oxygenation and perfusion.

- Provide initial supportive treatment while rapidly finding and treating the cause. Prepare for resuscitation and

intubation

- While cardiogenic shock is often fluid-resistant and there is high risk of pulmonary oedema, cautious IVF remains the first line treatment for compromised perfusion. Consider inotropes (LV failure), diuretics (fluid overload), nitrates (acute LV dysfunction with severe hypertension); NIPPV (severe persistent pulmonary oedema. Treat acute ischaemia (🕮 p. 110)
- Involve a cardiologist, if available, as soon as possible in all cases of cardiogenic shock

Disposition

Admit all patients with cardiogenic shock to the ICU (where available), review by a cardiac team ◈.

33 Tachycardia in adults

This chapter refers to the management of adults with a high HR (> 100) and a palpable pulse.

For the management of adults with tachycardia and no palpable pulse, see 🕮 p. 8. For the management of tachycardia in children, see 🕮 p. 92.

Considerations on rate

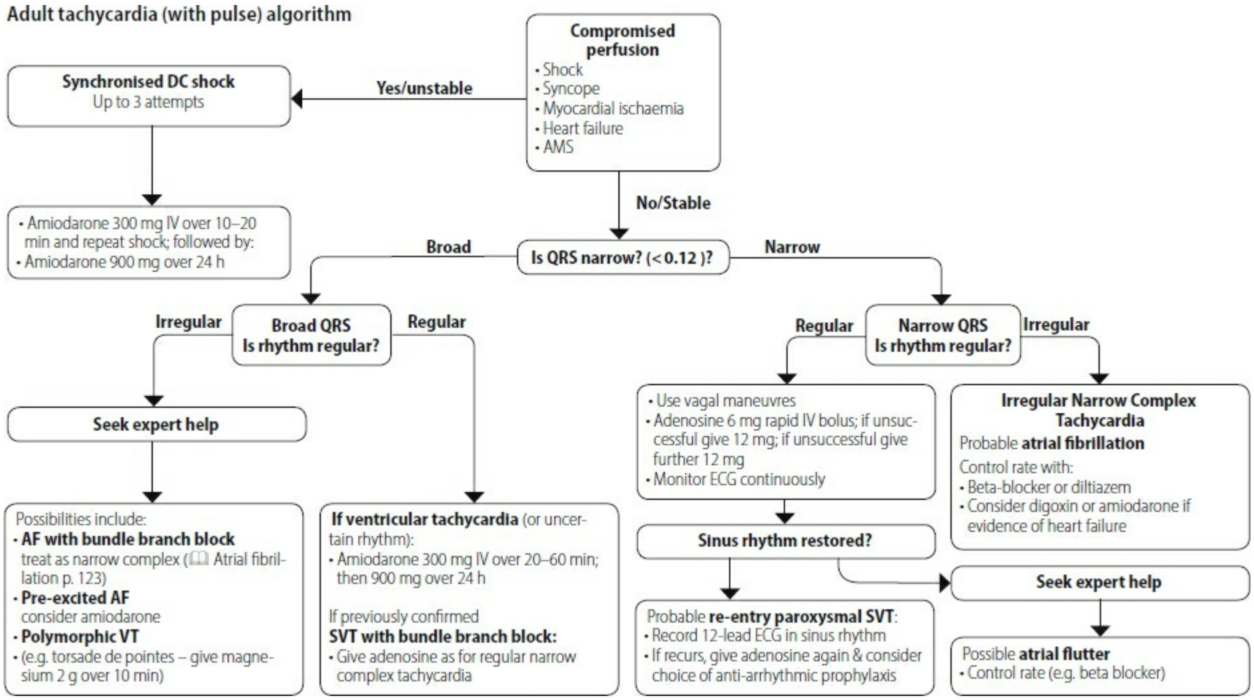
- Symptomatic tachycardia tends to involve a HR > 150
- HR > 180 in adults is likely due to supraventricular tachycardia
- While severe tachycardia can compromise filling and cause hypotension, a rate < 140 is unlikely to be the primary cause of hypotension. Also note that increased rate may be necessary to maintain cardiac output and compensate for underlying causes of shock such as sepsis or hypovolaemia. Always consider and treat other causes of poor perfusion; cardioversion will not help all unstable patients with tachycardia

General management

- Assess ABC
- Give O_2 if appropriate and obtain IV access
- Monitor ECG, BP, SpO2, record 12-lead ECG
- If no monitor, use the defibrillator's monitoring function
- If HR > 180 and no monitor, consider vagal manoeuvres
- We do not recommend drug treatment if no monitor available
- Identify and treat reversible causes (e.g. electrolyte abnormalities)

Torsade de Pointes

Torsade de Pointes is a polymorphic form of VT, associated with long QT syndrome, hypomagnesaemia and hypokalaemia that may degenerate to VF. The ECG tracing undulates as the QRS axis constantly changes. Treat with IV Mg, and replete K^+ to 4.5–5 mmol/l ◇. Recurrent episodes may require overdrive pacing ◈.



34 Tachycardia in children

This chapter refers to the management of children with a high HR with a palpable pulse.

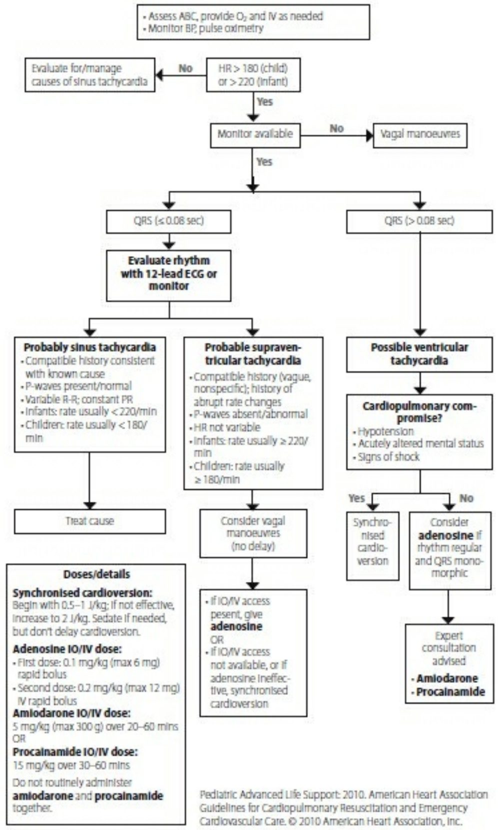
For the management of children with tachycardia and no palpable pulse, see 🕮 p. 18).

The definition of tachycardia in children varies dependent upon age, but is typically considered to be:

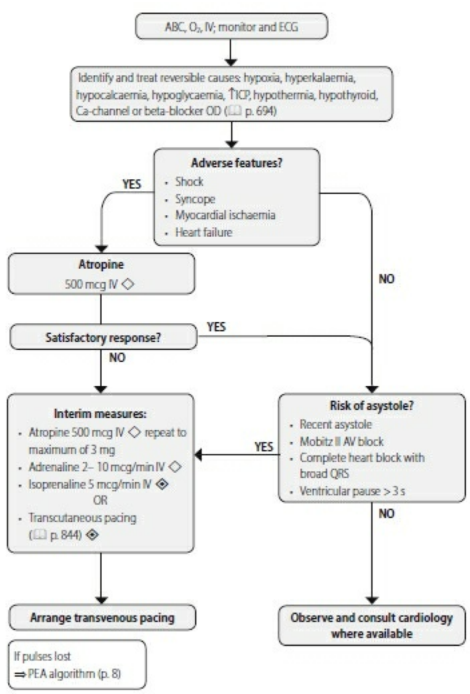
• Children – HR > 140 tachycardia, HR > 180 SVT
• Infants – HR > 180 tachycardia, HR > 220 SVT

Refer to the algorithm opposite.

• Vagal manoeuvres in infants or young children: place a plastic bag filled with ice and cold water over the face for 15 to 30 seconds, or stimulate the rectum with a thermometer. In older children, encourage Valsalva manoeuver for 15 to 20 seconds. Carotid massage and orbital pressure should not be performed in children.



35 Bradycardia



36 Mitral valve disease

Mitral valve (MV) disease is a major cause of morbidity and mortality. The commonest cause in Africa is rheumatic heart disease. Other causes include congenital abnormalities, cardiac tumours (stenosis), papillary muscle dysfunction, mitral valve prolapse, endocarditis and some drugs (incompetence).

The first five minutes

ABC, VS, O_2, pulse oximetry, IV access, cardiac monitor.

Mitral stenosis

Thickening and immobility of the MV leaflets causes an obstruction of blood flow from the left atrium (LA) to LV. This causes increased pressure within the LA, pulmonary vasculature and right side of the heart. Eventually LA dilatation and RV failure occur. Arrhythmias, particularly AF, are common.

History and physical examination

Patients usually present with a combination of palpitations (often in AF), chest pain, syncope and features of pulmonary hypertension (PHTN) and RVF.

Key historical features

Any embolic complications of AF, haemoptysis, or decreased effort tolerance. Ask about a history of rheumatic heart disease, features of pulmonary hypertension, and cardiac failure.

Signs and symptoms

Typically a combination of PHTN, RVF, AF and on auscultation.

- Loud S1 (if in sinus), opening snap (high-pitched early diastolic sound heard at or medial to apex during expiration), low-pitched rumbling mid-diastolic murmur at apex (heard best with patient in left lateral position)

Differential diagnosis

Severe aortic valve regurgitation, atrial septal defect, left atrial myxoma.

Investigations

Echo is the test of choice and can grade the severity of disease.

- ECG: ◇ (LA enlargement – wide (> 120 ms), notched P in lead II, deep (> 1 mm) negative portion of biphasic P-wave in V1 (if in sinus); AF; RAD; RVH)
- Imaging: CXR ◇ (straightening of left heart border, enlarged LA as double shadow of LA behind RA, prominent pulmonary arteries, Kerley B lines, dilated upper lobe veins); echo ◈ (most sensitive and specific method for detecting and judging the severity of disease; trans-oesophageal echo provides better detail than trans-thoracic)

Mitral regurgitation

History and physical examination

LV volume overload leads to LV dilatation and failure. Regurgitation results in LA dilatation and AF. Acute MR results in acutely raised LA and pulmonary pressures with acute LVF and pulmonary oedema.

Key historical features

- Acute: pulmonary oedema, hypotension, cardiogenic shock
- Chronic: asymptomatic for years then features of progressive LV and eventually RV failure. AF is relatively common

Signs and symptoms

Evidence of LVF, cardiac dilatation and possibly AF and RV dysfunction. Some classic findings include:

- Brisk carotid upstroke, laterally displaced hyper-dynamic apex beat, systolic thrill, soft S1, widely split S2, prominent low pitched S3, pansystolic high-pitched blowing murmur at apex and radiating to axilla

Differential diagnosis

VSD, aortic valve regurgitation.

Investigations

Echocardiography is the test of choice and can grade the severity of disease.

- ECG: ◇ (LA enlargement, LVH, atrial fibrillation)
- Imaging: CXR ◇ (cardiomegaly, LA and LV dilation, pulmonary congestion); echo ◈ (trans-thoracic – diagnose and quantify regurgitation, identify wall motion abnormalities; transosophageal – detailed information aids decision regarding definitive treatment)
- Cardiac catheterisation ◈: confirmation of severity and evaluation of valve or coronary disease

Management

The goal of acute management is to address hypoxia and hypotension and to treat arrhythmias. Severe mitral valve disease may include both stenosis and regurgitation, and often present with hypotension in the setting of dyspnoea. This is a difficult management challenge, as dyspnoea may result from severe pulmonary hypertension or pulmonary oedema. In the former case IVF is likely life-saving; in the latter, IVF may be life-threatening.

Mitral stenosis

Medical

- SBE prophylaxis, treat RVF, treat AF, refer to cardiology for consideration for percutaneous intervention (balloon valvotomy) or valve replacement

Surgical

- Open valvotomy: where balloon valvotomy is not available or appropriate
- MV replacement: patients with significant regurgitation or severe disease

Mitral regurgitation

Outcome is poor in severe symptomatic regurgitation without surgical intervention.

- Mild symptoms: decrease afterload with vasodilators ◇ (hydralazine or ACE-I) (increases cardiac output and reduces regurgitation). AF – rate control and anticoagulation
- Acute decompensation: treat pulmonary oedema with diuretics ◇, mechanical ventilation ◈; blood cultures × 3 ◇ . Afterload reduction with nitrates ◇

Disposition

Admit mitral regurgitation patients with new or worsening DIB or poor perfusion.

Discharge patients with mild stenosis (MV area > 1.5 cm^2) and mild symptoms, for follow-up at outpatient specialist clinic. Refer all symptomatic patients.

37 Other structural valve disease

Consider valve disease in all patients with syncope, chest pain, heart failure, and murmurs. In Africa most is due to rheumatic heart disease (RHD); other causes include congenital abnormalities, infective endocarditis, and age-related changes. If there are RHD-associated aortic or tricuspid abnormalities, mitral valve disease is almost always present (📖 p. 95).

The first five minutes

- ABC, VS, O_2, IV, pulse oximetry, cardiac monitor
- Bedside cardiac US
- ECG

General approach

Examination

Features of cardiac failure, arrhythmias, adequacy of perfusion, pulses and JVP, ventricular dilatation and/or hypertrophy, murmurs and fever.

Diagnostic studies

- ECG: arrhythmias, conduction abnormalities, ventricular and/or atrial enlargement, ischaemia.
- Imaging: CXR: abnormal cardiac size and shape, evidence of cardiac failure.

Arrange for echo for patients with:
- Any murmur associated with symptoms or an abnormal ECG or CXR
- All diastolic, continuous, holosystolic, late systolic murmurs, and murmurs with ejection clicks or with radiation to the neck or back

Aortic stenosis

History and physical examination

Patients are asymptomatic when the valve area remains sufficiently large. Over time, valve area narrows causing angina-like chest pain, syncope, or CCF.

A mid-systolic ejection murmur with radiation to the neck is present, heard best over the right-upper sternal border. The aortic component of S2 may be soft or absent. Pulse pressure is narrow and arterial pulses may have a slow upstroke.

Management

There is no effective medical therapy for aortic stenosis. Surgical intervention ◈ is indicated for symptoms. In patients critically ill from aortic stenosis:
- Cautiously administer fluids while avoiding pulmonary oedema
- Ensure sinus rhythm
- Avoid vasodilators and diuretics
- Avoid beta-blockers in symptomatic patients
- Symptomatic management of chest pain and dyspnoea

Chronic aortic insufficiency

History and physical examination

Symptoms range from none to CCF with volume overload.

On examination an early-diastolic murmur is heard best at the left lower sternal border, with lateral displacement of the apex beat. Pulse pressure is wide, and collapsing peripheral pulses may be found.

Management

- If symptomatic, provide a vasodilator ◇ (nifedipine or hydralazine) to augment forward flow and reduce regurgitant volume

- Surgical intervention ◈ is indicated in severe aortic insufficiency if symptoms or LV dysfunction are present

Acute aortic insufficiency

History and physical examination

- The patient will be acutely ill with features of acute LVF (tachycardia, cardiogenic shock, and pulmonary oedema). MI and sudden death are common
- The classic findings of chronic AI may be absent. The diastolic murmur will be short and soft or not heard at all, and pulse pressure may be normal

Investigation

- Echo ◈ is critical in confirming the diagnosis

Management

Cautious vasodilators and diuretics ◇ to enhance forward flow and optimise volume status. Arrange for emergency surgical intervention ◈.

Tricuspid valve disease

Tricuspid regurgitation is more common, but tricuspid stenosis can be caused by RHD. In the majority of cases, the symptoms and exam findings of mitral and aortic disease will predominate. No specific medical therapies.

Pulmonary valve disease

Pulmonary stenosis predominates, and is usually congenital or due to RHD. Clinical features are those of RVF (dyspnoea, fatigue, exertional syncope, chest pain, oedema). No specific medical therapies, and balloon valvotomy ◈ indicated in symptomatic patients.

38 Infective endocarditis

Infective endocarditis is infection of the endocardium that may involve valves or the mural endocardium. The most common cause is bacterial (usually *Streptococcus*; *Staphylococcus aureus* seen) though infection by viruses, fungi and rickettsia may occur. Non-infective inflammatory endocarditis is a rare complication of some auto-immune diseases (SLE) and malignancies.

Clinical presentation varies widely, from subtle signs and vague constitutional symptoms to acute heart failure and sepsis. Definitive diagnosis may be challenging and a prolonged course of IV antibiotics is needed for effective treatment.

IE is divided into two types:

- Native valve endocarditis:
  - » Acute – aggressive type that affects both normal and abnormal valves
  - » Sub-acute – more common in abnormal valves
- Prosthetic valve endocarditis:
  - » Early – occur within 60 days of surgery; mostly iatrogenic
  - » Late – beyond 60 days of surgery; community acquired, haematogenous

The first five minutes

- ABC, VS, O_2, pulse oximtery, IVF, cardiac monitor
- Bedside cardiac US

History and physical examination

Key historical features

Identify pre-existing risk factors (almost any structural cardiac and valvular disease; IV drug abuse; immune compromise; recent invasive procedure).

Signs and symptoms

Look for evidence of systemic infection (fever – most common feature, malaise, constitutional symptoms, weight loss), cardiac involvement (palpitations, syncopy and chest pain, arrhythmias, failure and murmurs), immune phenomena (splenomegaly, nephritis, Osler nodes, Roth spots) and vascular phenomena (Janeway lesions, systemic emboli and bleeding).

- Acute illness: rapid onset, quick development of complications and deterioration (associated with highly virulent *Staphylococcus*)
- Sub-acute illness: an indolent course but can result in valve damage and septic emboli

Investigations

- Labs: CBC, electrolytes, renal, urinalysis; blood culture ESR ◇
- ECG ◇ (tachycardia, conduction abnormalities, arrhythmia)
- Echo: ◈ (essential part of diagnosis; trans-oesophageal much more sensitive than trans-thoracic; look for vegetations, paravalvular abscess and leaks, new regurgitation)

Diagnostic criteria

Diagnosis may be presumed with:

1 Two positive blood cultures from two different sites AND
2 Echo evidence of endocarditis, e.g. vegetation, paravalvular abscess, new regurgitation, immune phenomena, vascular phenomena.

Duke criteria for IE:

- Major criteria:
  - » Positive blood culture for IE
  - » Evidence of endocardial involvement
- Minor criteria:
  - » Predisposition
  - » Fever
  - » Vascular phenomena
  - » Immunologic phenomena
  - » Microbiological evidence
  - » Echocardiographic findings

The full criteria can be found at: http://reference.medscape.com/calculator/endocarditis-diagnostic-criteria-duke.

Management

The goal of acute management is early identification and treatment of complications, and aggressive antibiotic therapy.

- Manage complications: CCF, septic shock or emboli (📖 Congestive cardiac failure, p. 104 and Toxic shock, p. 380)
- Antibiotics: start empiric IV antibiotics after drawing the culture samples. Refer to your local guidelines for specific treatment regimes. Two weeks of combination therapy using penicillin with an aminoglycoside is effective in most native valve cases. Alternative combinations include ceftriaxone and an aminoglycoside. Consult for prosthetic valve
- Continue for at least four weeks

Disposition

Admit all patients for IV therapy; involve cardiology early.

39 Cardiac tamponade

Cardiac tamponade occurs when cardiac filling and contraction are impaired by a pericardial effusion (including haemopericardium). This results in decreased cardiac output that can rapidly lead to shock, and ultimately death, if not promptly treated.

The first five minutes

- ABC, VS, O_2, IVF, blood for investigation, cardiac monitor
- ECG
- Bedside cardiac US
- Prepare for pericardiocentesis and possible intubation

History and physical examination

Key historical features

Prior diagnosis or symptoms of pericardial effusion: pleuritic chest pain, palpitations, malaise. As the effusion volume and pressure increases it may cause decreased effort tolerance, dyspnoea and ultimately collapse. Any history of trauma, malignancy or renal failure.

Signs and symptoms

- Tamponade can be extremely challenging to diagnose. Clinical findings are notoriously inconsistent. US, if available, is extremely useful
- Beck's triad – hypotension, elevated JVP, muffled heart sounds – is classic but rarely complete. Tachycardia, distended neck veins, pulsus paradoxus (decrease in pulse volume greater than 10 mmHg with inspiration) and features of cardiac failure may be present. The patient may present with shock and circulatory collapse

Possible causes and differential diagnosis

Possible causes: any cause of pericardial effusion may lead to tamponade. The most common causes are trauma, viral pericarditis, TB, malignancy, renal failure, and thoracic surgery.
Differential diagnosis: any cause of shock or acute cardiac failure, tension PTX, pulmonary embolism, myocarditis, acute coronary syndromes and constrictive pericarditis.

Investigations

Tamponade is a clinical diagnosis, possibly assisted by bedside US. These patients are usually too sick and unstable to tolerate formal investigation.
- ECG: ◇ (tachycardia, low voltage complexes, electrical alternans)
- Imaging: CXR ◇ (may show enlarged cardiac silhouette); bedside US (investigation of choice), formal echocardiography ◈ (pericardial effusion with right ventricular or right atrial collapse)

Management

The goal of acute management is to restore cardiac filling and output.

Unstable patient

- Give 1 l IV crystalloid bolus to increase right sided filling pressures while preparing the patient for pericardiocentesis
- Perform emergency pericardiocentesis: US guided is preferred. (📖 Pericardiocentesis, p. 842).

Disposition

Admit all patients with tamponade for further intervention and review by cardiac team; immediate cardiothoracic or general surgery consultation for all traumatic cases.

40 Congestive cardiac failure

Congestive cardiac failure occurs when the heart cannot maintain adequate cardiac output. This may be due to inadequate filling (diastolic failure) or inadequate emptying (systolic failure) of the ventricles. Failure of one ventricle may dominate, or it may be biventricular.

The most common causes worldwide are hypertension, diabetes mellitus, ischaemic heart disease, valvular heart disease, cardiomyopathies, pregnancy-induced cardiomyopathy and congenital heart disease. In Africa, rheumatic valvular disease and late presenting congenital heart disease are common causes.

The first five minutes

- ABC, upright position, O_2, IV access, cardiac monitor
- Management varies, but strongly consider early nitrates, diuretics and CPAP

History and physical examination

The clinical evaluation should aim to answer three critical questions:

1. Is cardiac failure present and is it mainly left, right, or both ventricles?
2. What is the underlying cause?
3. What has precipitated this decompensation?

Key historical features

The cardinal symptoms of CCF are decreased effort tolerance, dyspnoea, oedema, palpitations and syncope/dizziness.

Signs and symptoms

- Left: decreased effort tolerance, dyspnoea, orthopnoea, paroxysmal nocturnal dyspnoea, dizziness and syncope, palpitations, chest pain, displaced apex beat, gallop rhythm
- Right: oedema, ascites, pleural effusion, increased JVP, hepatic congestion
- Aetiologic features: chest pain, palpitations, hypertension, hyperglycaemia, arrhythmias, murmurs, fever, features of RHD, features of SIBE
- Precipitating factors: infection/sepsis, anaemia, ischaemia

Differential diagnosis

Any cause of dyspnoea/shock (bronchospasm, pneumonia, sepsis, anaphylaxis, PE, PTX etc.) and any cause of oedema/fluid overload (renal/hepatic failure, hypoproteinemic states, lymphatic disease).

Investigations

Most useful initial investigations are an ECG, CXR and bedside US if available. Remember pregnancy test in women; Hgb in all patients.

- Labs: CBC, renal, electrolytes ◇; consider cardiac enzymes, thyroid function tests ◈
- ECG: ◇ (may reveal aetiology, i.e. ischaemia, arrhythmia, hypertrophy)
- Imaging: CXR ◇ (cardiomegaly, pleural effusion, upper lobe blood diversion, Kerley lines, peri-bronchial cuffing); echo ◈ (definitive information on structure, function and often aetiology)

Management

The goal of acute management is to support oxygenation and restore circulatory status while searching for and

treating the cause of the CCF and the precipitant of this decompensation.

- Nitrates ◇: IV 5–10 mcg/kg or infusion or SL (0.4 mg Q5min) for all patients with LVF, pulmonary oedema and a SBP > 90–100 mmHg. Especially useful in ischaemia and acute severe hypertension. Caution with RV infarcts.
- Diuretics (IV furosemide) ◇: for all cases of RHF. Also in LVF if initial nitrate treatment is not effective or appropriate, or if the patient is clearly fluid overloaded 40–100 mg IV (consider higher doses in patients already taking or in those with renal failure)
- NIPPV (non-invasive positive pressure ventilation) ⟐: if the patient is in respiratory distress, place the patient on BiPAP or CPAP
- Morphine ◇: low doses (1–2 mg) may help air hunger; its use is controversial; avoid in CCF following acute MI
- Although counter intuitive, patients with acute LV failure, especially with hypertension, often require a small IV fluid bolus as adjunct to nitrate therapy
- Consider cautious transfusion in patients with a very low Hgb

Cardiogenic shock

See 🕮 p. 88.

Disposition

Admit all patients with acute severe heart failure or first presentation of CCF. Observe patients with mild- to moderate decompensations of chronic CCF for six hours.

41 Severe hypertension

Hypertension is a chronic condition associated with end-organ damage and acute complications. Severe hypertension with accelerated end organ damage is associated with a high risk of death.

'Hypertensive urgency' is often used to describe severe hypertension without evidence of acute end organ damage, while 'Hypertensive emergency' implies severe hypertension with evidence of acute end organ damage.

Acute end organ damage:

- Cardiac (MI, aortic dissection, acute LVF)
- Renal (acute kidney injury)
- Brain (encephalopathy, stroke, SAH)
- Placenta (eclampsia)

The first five minutes

- ABC, VS, O_2, IV, SaO_2, cardiac monitor
- ECG

History and physical examination

Key historical features

Known hypertension, chronic end organ damage and medication. Stimulant and alcohol use. Ask about acute end organ damage (chest pain, dyspnoea, headache, confusion, neurological abnormalities, visual disturbance), pregnancy.

Signs and symptoms

Evidence of end organ damage may include:

CNS – AMS, focal neurological deficits; cardiac – tachycardia, CCF, shock, pulse deficits (AD); abdomen – palpable pregnancy, AAA. Also perform fundoscopy, looking for papilloedema and retinal bleeds.

Investigation

- Labs: CBC, electrolytes, renal function, urinalysis ◇; cardiac enzymes, PT/PTT ◈
- ECG ◇ (ischaemia, dysrhythmias, LVH)
- Imaging: CXR ◇ (pulmonary congestion or widening of mediastinum); echo (wall motion abnormality), CT head (CVA) ◈

Management

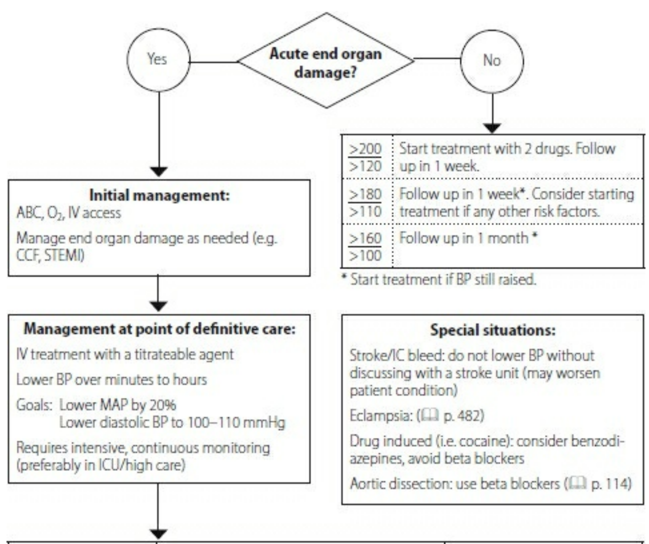
The goal of acute management is to reduce BP and avoid complications.

- Patients without acute end organ damage: start on single oral treatment (observe for two hours after first dose)
- Patients with evidence of acute end organ damage require acute BP control with titratable IV agents. Aim for a 10–20% reduction of MAP over a few hours. See Common emergency medications (📖 p. 957). Aortic dissection and intracranial haemorrhage are exceptions (📖 p. 468 and p. 472)

Refer to the accompanying flow diagram for detail.

Disposition

Admit all patients with evidence of acute end organ damage. Discharge patients without acute end organ damage for primary care follow up.



Nitroglycerin ◇	Infusion: start 5 µg/min q3–5 min to 20 µg; if no response, increase by 10 µg/min q3–5 min, up to 200 µg/min	Good for ACS, CCF, pulmonary oedema
Labetalol ◈	20 mg IV over 2 min. May administer 40–80 mg q10 min to max total: 300 mg. Infusion: 2 mg/min, titrate to response up to max total dose: 300 mg	Good for aortic dissection, neurological and renal complications, eclampsia
Nitroprusside ◈	Infusion: 0.3-0.5 µg/kg/min. Increase in 0.5 µg/kg/min increments. Titrate to effect.	Good for HPT encephalopathy, aortic dissection (must use with beta blocker)
Hydralazine ◈	Slow IV or infusion: 5 mg initially. Repeated doses of 5-10 mg q10 min as needed.	Second line for eclampsia. Beware precipitous BP drop

42 Pulmonary hypertension

Pulmonary hypertension (PHTN) is characterised by progressive and sustained increase in pulmonary vascular resistance that eventually leads to RV failure. It may be primary (unknown aetiology) or secondary.

WHO Classification

- Group 1: primary pulmonary artery disorder

- Group 2: due to left heart failure
- Group 3: due to pulmonary disorders
- Group 4: due to thromboembolic occlusion of the proximal or distal pulmonary vasculature
- Group 5: due to haematologic, metabolic, and other systemic disorders

The first five minutes

- ABC, VS, O_2, IV, pulse oximeter, cardiac monitor
- ECG

History and physical examination

Clinical features include:
1. Primary features of PH
2. Features of RV pressure overload and failure
3. Features of the causal condition

Key historical features

Chest pain, effort intolerance, dyspnoea, palpitations, dizziness, syncopy. Symptoms of right heart failure, distended neck veins, pulsatile hepatomegaly, extremity oedema. Hoarseness caused by compression of the recurrent laryngeal nerve by the distended pulmonary artery is a rare symptom.

Signs and symptoms

RV hypertrophy (sternal heave, right sided S4, loud P2, widely split S2, functional pulmonary stenosis murmur). RV failure (oedema, raised JVP, hepatomegaly, right sided S3, ascites and pleural effusions). Arrhythmias. Search for the cause (e.g. wheeze with COPD, crackles with bronchiectasis). Acute PHTN caused by massive PE presents as shock with chest pain and dyspnoea.

Differential diagnosis

Any cause of RV hypertrophy or failure. Consider other causes of chest pain and dyspnoea.

Investigations

Echocardiography and cardiac catheterisation are the most useful diagnostic tests. Other investigations should be tailored to the suspected aetiology.
- Labs: CBC, ABG, HIV, urinalysis; PT/PTT, cardiac enzymes ◈
- ECG: ◇ (RVH, RAD, RBBB, RT enlargement)
- Imaging: CXR ◇ (enlargement of central pulmonary arteries, enhancement of the peripheral vessels; may show primary lung pathology such as bronchiectasis); echo ◈ (confirm diagnosis, assess severity, identify cause)
- Right heart catheterisation ◈ (most precise estimate, PHTN is confirmed when mean pulmonary artery pressure is ≥ 25 mmHg at rest)

Management

The goal of acute management is treatment of RV dysfunction and maintaining haemodynamics.
- Furosemide ◇ diuresis in patients with fluid retention. Diurese carefully as may not tolerate preload reduction well
- Patients with severe primary PHT tolerate infection poorly: early and aggressive antibiotics
- Manage any arrhythmias as usual (🕮 Tachycardia, p. 90 and Bradycardia, p.94)
- Consider thrombolysis or thrombectomy ◈ for suspected massive PE
- Chronic therapy for patients with persistent PHTN WHO class II, III, or IV after treatment for the underlying cause

Disposition

Admit after stabilisation.

43 Ischaemic heart disease: acute coronary syndrome

Ischaemic heart disease (IHD) results from inadequate blood supply to the myocardium due to coronary artery stenosis, and is an increasing cause of death and disability in Africa. The most common cause is atherosclerotic coronary artery disease. Risk factors include male gender, increasing age, hypertension, diabetes mellitus, smoking and dyslipidaemia.

Chronic IHD can lead to CCF and arrhythmias. Acute coronary artery occlusion causes acute coronary syndromes (ACS) – unstable angina, non-ST elevation myocardial infarction (NSTEMI) and ST elevation myocardial infarction (STEMI). ACS is associated with significant morbidity and mortality.

The first five minutes

- ABC, VS, O_2 (keep sats > 93%) IV pulse oximeter, cardiac monitor
- ECG
- Aspirin (150 mg, chewed), nitroglycerin (avoid if inferior MI or low BP), IV morphine if needed and BP OK

History and physical examination

Key historical features

Ask about prior ACS, or cardiac intervention and risk factors. Classic ACS chest pain, pressing or squeezing, worsened by exertion, relieved by rest/nitrates, may spread to the arms or jaw, may be associated with diaphoresis, nausea, angor animi, dyspnoea and palpitations. Other symptoms (e.g. syncope, dyspnoea, confusion) can be considered ‘anginal equivalents’.

Angina is ‘unstable’ if it is of new onset, starts at rest, increases significantly in duration frequency or intensity, or is not relieved by rest and/or nitrates.

Signs and symptoms

Timing, duration, trigger, location, evolution of pain. Nausea, vomiting, diaphoresis, dizziness, weakness, syncope. VS: hypotension, tachycardia and bradycardia indicate severe disease. Cardiac: features of CCF, arrhythmias and murmurs.

Differential diagnosis

AD, PE, PTX, myocarditis, pericarditis, oesophageal spasm or rupture, peptic ulcer disease, costochondritis.

Investigations

- Labs: Hgb (anaemia), electrolytes, glucose, renal ◇; cardiac enzymes (troponin is the most sensitive and specific, is detectable at three hours, and remains elevated for seven days; CK-MB less specific, is detectable at three hours, peaks at 20–24 hours, and normalises after 2–3 days) ◈
- ECG: ◇ (serial ECGs essential; look for features below; dynamic change especially important)
- Imaging: CXR ◇ (mediastinal widening to suggest aortic dissection, cardiomegaly, pulmonary oedema, or PTX), bedside echo ◇; coronary artery CT ◈
- Angiography (with or without intervention)

ECG features of ischaemia

- Unstable angina, NSTEMI: peaked T-waves, ST depression (> 0.5 mm), dynamic T-wave inversion, arrhythmias and conduction abnormalities
- STEMI: ST elevation of > 1 mm in two or more anatomically contiguous leads; new LBBB; reciprocal ST

depression (📖 ECG, p. 84)
• Established infarction: pathological Q-waves

Management

The goal of acute management is to restore coronary perfusion and prevent complications.

• ABC, O_2 – target normal O_2 saturation
• Aspirin 150–300 mg (or clopidogrel if true aspirin allergy)
• Nitroglycerin 0.4 mg SL every 5 minutes if SBP > 100. IV nitrates for resistant pain or ACS with pulmonary oedema
• IV morphine for continued pain; avoid high doses; ongoing pain should prompt urgent treatment of ongoing ischaemia
• For percutaneous coronary intervention (PCI) ◈ confirmed diagnosis of STEMI preferred. If PCI not available in < 120 minutes, treat with thrombolysis (alteplase or streptokinase (1.5 million units over 1 hour)). Ensure critical care unit available after thrombolysis
• Antiplatelet therapy ◈: clopidogrel for patients treated with thrombolysis; for PCI, consult cardiology
• Anticoagulation: all patients with UA or MI should receive anticoagulation. Unfractionated heparin best for patients undergoing PCI or medical management. Low molecular weight heparin (enoxaparin) and bivalirudin are other options

Disposition

• Admit all patients with ACS
• STEMI – transfer to centre with PCI ◈ if can be done in < 2 hours from symptom onset. If > 2 h and < 12 hours, give thrombolytics. Admit to ICU

44 Abdominal aortic aneurysm

Abdominal aortic aneurysm (AAA) is an abnormal focal, full thickness dilatation of the abdominal aorta due to a weakened vessel wall. It is associated with significant morbidity and mortality, particularly when associated with an acute symptomatic presentation. The four dangers of AAA are:

1. Rupture with massive haemorrhage and death
2. Dissection with occlusion of arterial supply to organs (i.e. mesenteric or renal infarction)
3. Thrombosis with aortic occlusion or embolism causing limb or organ ischaemia
4. Local mass effects

The greater the dilatation the greater the risk of rupture. Acute symptomatic AAA usually represents leak or rupture.

The first five minutes.

• ABC, O_2, IVF (via 16G or larger), blood for investigation and cross match, cardiac monitor
• Bedside US

History and physical examination

Important historical features

Patients are often asymptomatic. Consider symptoms related to embolic phenomena (limb ischaemia, mesenteric ischaemia), arterial dissection (as above) and local effects (abdominal, pelvic or back pain).

Risk factors for AAA

Age, male gender, smoking, hypertension, hypercholesterolaemia, diabetes mellitus, a family history of atherosclerotic vascular disease, established atherosclerotic disease (CAD, PVD), CAD equivalents, or other vascular inflammatory conditions (RA, SLE).

Signs and symptoms

AAA rupture has an immediate risk of death in excess of 65%. Those who survive to hospital present with shock, abdominal pain and possibly a pulsatile abdominal mass. Evaluate haemodynamic status and search for anaemia. The abdomen may be tender. Carefully evaluate distal pulses. The classic triad of abdominal pain, abdominal distension and hypotension is present in only 50% of patients with ruptured AAA. Bedside US has good sensitivity and specificity for the detection of AAA.

Investigations

- Labs: CBC, electrolytes, renal, ABG, type and cross ◇; PT/PTT, lactate, cardiac enzymes ◈
- ECG: ◇ (normal to signs of ischaemia)
- Imaging: abdominal US ◇ (high sensitivity and specificity); abdominal CT ◈ (can evaluate for rupture or active leak)

Management

The goal of acute management is stabilisation and surgical repair.

Symptomatic patients require rapid evaluation, resuscitation and the involvement of a vascular surgeon. Control pain with IV morphine or fentanyl. Establish good vascular access and order blood and blood products early.

In unstable patients with suspected rupture resuscitate with fluids and blood products. Aim to keep systolic BP around 90–100 mmHg to reduce shearing forces in the aneurysm.

When AAA is an incidental finding in asymptomatic patients, it may be followed with serial exams, aggressive vascular risk factor control and elective repair based on size; dilatation above 4 cm – 5% per year risk of rupture; above 5 cm – 15% (consider repair); above 8 cm – 50% (needs repair).

Critical documentation

Document VS, examination and US findings; response to fluids and vasopressors (if used). Record time of call to surgical consult, and time of arrival.

Disposition

Admit symptomatic patients for surgery; most will be disposed directly to the OT.

45 Aortic dissection

Aortic dissection (AD) occurs when a tear in the intima allows blood to enter the space between the intima and media of the aortic wall, creating a false lumen. This may: erode through the wall causing aortic rupture; dissect back into the true lumen, or extend down the aorta causing occlusion of major branches and ischaemia in their distribution. AD may also progress to the aortic root, causing acute aortic stenosis or incompetence and coronary ischaemia.

It is classified using either the Stanford or DeBakey system, with the key fact being whether the ascending aorta is involved (usually requires urgent surgical management). AD usually involves the thoracic aorta though isolated abdominal dissecions do occur and are associated with existing AAA in 40% of the time.

The Stanford classification system:

- Stanford A – dissection involving the ascending aorta with or without involvement of the descending aorta
- Stanford B – dissection sparing the ascending aorta

The first five minutes

- ABC, VS, O_2, IV, IVF, type and cross, cardiac monitor

• Involve the surgical team from the outset

History and physical examination

AD presents with chest or abdominal pain and a wide range of associated features, depending on the location. Diagnosis can be extremely difficult.

Key historical features

The primary risk factor is hypertension. Other predisposing conditions include atherosclerosis, connective tissue disease (Marfan's), congenital abnormalities of the heart and aorta (coarctation) and stimulant abuse (cocaine).

Signs and symptoms

Chest pain is present in more than 90% of patients, often described as sharp, tearing and maximum at onset. Other clinical features are associated with the progression of the dissection: shock (rupture), cardiac failure (involvement of the aortic root or coronary arteries), stroke (involvement of the carotid arteries), upper limb ischaemia (subclavian arteries), spinal artery dissection with acute spinal cord syndromes, abdominal pain (visceral or mesenteric ischaemia).

Investigations

- Labs: CBC, renal, type and cross ◇; PT/PTT, cardiac enzymes ◈
- ECG: ◇ (findings range from normal to signs of ischaemia)
- Imaging: CXR ◇ (insensitive but may show widening of mediastinum, indistinct aortic knob, double knuckle sign, trachea deviation towards right, a pleural cap, pleural effusion or may be normal (up to 12%)); echo ◈ (transthoracic: low sensitivity and specificity; transoesophageal: highly sensitive and specific); CT-angiography ◈ (provide definitive diagnosis, modality of choice if available)

Management

The goal of acute management is to reduce shearing forces and limit extension.

Reduce BP and control HR: aim for SBP 100–120 mmHg and HR 60–80. Manage with intra-arterial BP when possible.

Medical management is the initial treatment of choice for Stanford B; emergency surgery for type A, and any type B that fails medical management.

- Analgesia: IV morphine or fentanyl ◇
- BP control: IV, titratable beta-blockers are the cornerstone of therapy. Add a vasodilator if needed, but do not use vasodilators alone as reflex tachycardia (and increased shear forces) may result
- Beta-blockers ◇, in order of preference: esmolol 500 mcg/kg as bolus then infusion of 50–200 mcg/kg/min. Labetalol 20 mg bolus, then every 5–10 min (both contraindicated in bradycardia, COPD and hypotension
- Vasodilators: nitroprusside ◇ (0.5–3 mcg/kg/min) started AFTER beta-blockers
- Calcium channel blockers (with exception of nifedipine) if beta-blockers contraindicated

Critical documentation

Document clinical findings, investigations and results, medical therapy and response, and time of consultation.

Disposition

Admit all patients, preferably to ICU ◈.

46 Deep vein thrombosis (DVT)

DVT is the abnormal formation of a clot in the deep venous system. It can occur anywhere but is most frequent in the lower limbs and pelvis. The clot may embolise (possibly leading to PE) or cause local obstructive effects (venous insufficiency syndromes). Prompt recognition and management is essential to prevent dangerous

complications, primarily PE.

The first five minutes

Patients with DVT are often well. If ill-appearing:

- ABC, O_2, IV, cardiac monitor
- Rapidly investigate, focus on evaluation for PE (📖 p. 118)

Risk factors

- History of immobilisation or prolonged, hospitalisation/bed rest
- Recent surgery, lower extremity trauma
- Obesity
- Prior history of DVT or other clotting disorder
- Malignancy
- Use of oral contraceptives or hormone replacement therapy
- Pregnancy or postpartum status
- Stroke
- Central venous catheter

History and physical examination

Key historical features

Unilateral limb pain and swelling. May also complain of leg 'heaviness', pruritus or redness. Ask about DVT risk factors.

Signs and symptoms

Unilateral limb swelling (compare sides) and oedema. Look for features of chronic venous insufficiency. Erythema and superficial vein distension possible. Always evaluate adequacy of arterial perfusion and consider signs of infection. Search for features of a possible cause such as pregnancy and pelvic or abdominal masses. Search for evidence of PE (tachycardia, tachypnoea, hypoxia, effusion).

Critical: venous infarction (*phlegmasia cerulea dolens*) presents with severe leg pain and swelling, with cyanosis – typically leads to large pulmonary embolus; prompt recognition and treatment is essential.

Differential diagnosis

Cellulitis, trauma, venous insufficiency, superficial thrombophlebitis, lymphangitis or lymph obstruction, Popliteal (Baker's) cyst, rare causes (osteomyelitis, malignancy etc.)

Investigations

Investigations should be guided by risk assessment, using a tool like the Well's DVT criteria.

- Labs: pregnancy test ◇; D-dimer ◈ (not useful in inpatients, pregnancy beyond first trimester, patients with known malignancy)
  For low risk patients, a negative D-dimer rules out DVT. For intermediate to high risk patients, and those with a positive D-dimer, investigate formally
- Imaging: compression US ◇; formal Doppler US, CT venography, formal venography ◈

Management

The goal of acute management is to confirm the diagnosis and exclude PE.

Well patients with DVT require anticoagulation and identification and reversal of the cause.

Anticoagulation: initially with unfractioned heparin or LMWH, bridging to oral anticoagulants such as warfarin (📖 p. 959).

More invasive therapies like thrombolysis, thrombectomy and IVC filters should be guided by a specialist.

Disposition

Admit patients being treated with unfractionated heparin, with increased risk of bleeding and those with co-morbidities.

47 Pulmonary embolism

Pulmonary embolism (PE) is the formation or deposition of thrombus in the pulmonary circulation and may be asymptomatic or rapidly fatal. Larger PE may cause two dangerous physiological effects: ventilation-perfusion (VQ) mismatch creating dead space ventilation and hypoxia, or sudden RV strain with acute RV failure, resulting in acutely reduced pre-load, LV failure and poor cardiac output.

Diagnosis can be extremely difficult. Symptoms are non-specific and investigations are often unhelpful. Advanced imaging such as CT angiography and VQ scanning are expensive and often not available. Well-studied decision rules are available, though there is limited validation in this regional context. In addition, decisions about diagnostic testing for PE must be made with consideration of potential therapies. For example, in patients who have contraindications to anticoagulation or in areas where thrombolytics and anticoagulants are not available, a confirmed diagnosis of PE may have limited impact on management.

The first five minutes

- ABC, VS, IV, pulse oximetry, cardiac monitor
- ECG

History and physical examination

Key historical features

Sudden onset dyspnoea (commonest symptom), pleuritic chest pain, cough ± blood, sweats, syncope, apprehension (feeling of impending doom).

Signs and symptoms

No single or group of findings are sensitive or specific for PE. Common features include:

- Tachypnoea (most common), tachycardia, wheeze, cough
- Low grade fever (37–38°C)
- Clinical evidence of DVT
- Hypotension
- Signs of right heart failure (only with large PEs, or multiple longstanding PEs):
  - » Loud and/or wide splitting of S2
  - » RV gallop and heave
  - » Elevated JVP with prominent a waves
  - » Pulmonary incompetence murmur

> Pre-test probability is best calculated using simplified Well's score:
> - Signs/symptoms of DVT: 3 points
> - PE is the most likely diagnosis (clinically): 3 points
> - Tachycardia (> 100 b/min): 1.5 points
> - Past history of DVT or PE: 1.5 points
> - Immobilisation or surgery within last 4 weeks: 1.5 points

• Haemoptysis: 1 point
• Active malignancy within last 6 months: 1 point
< 2 Low pre-test probability
2–6 Intermediate pre-test probability
> 6 High pre-test probability

Investigations

In many settings, once there is clinical suspicion for PE, decision-support tools such as Well's PE criteria are used to stratify risk and direct diagnostic testing. There is, however, no regional validation of these criteria and they may not perform well in settings where relative prevalence of potential risk factors for hypercoagulability (e.g. HIV and HIV-therapies) are different from settings where the tools have been validated. We have reproduced Well's criteria to give a sense of high-risk factors that may be integrated with other clinical evaluation, but do not endorse their use as a primary tool for management in the absence of regional validation.

- Labs: PT/PTT, D-dimer (very high specificity in a patient with very low risk; positive result in any risk group or a negative result in moderate to high patients has little diagnostic value), troponin (elevated in large PEs and useful as prognostic indicator) ⟐
- ECG: ◇ (tachycardia common; often normal or non-specific ST/T changes)
- Imaging: CXR ◇ (abnormal in ~75%; usually non-diagnostic – may show plural effusion, infarcted segment or a 'Hampton's hump'; may confirm alternate diagnoses); V/Q scan ⟐ often inconclusive, safe in pregnancy (interpret with clinical and CXR findings 5%); CT angiogram ⟐ esp. if underlying lung disease; negative test does not exclude smaller PE in high risk patients), echo ⟐ (useful for risk assessment)

Management

The goal of acute management is to restore oxygenation and cardiac output.
Provide O_2, IVF and inotropes as needed.

- Anticoagulation: enoxaparin or unfractionated heparin to bridge to warfarin
- Thrombolysis only in massive PE with clinical instability and cardiogenic shock. Not indicated in the absence of RV failure

Disposition

Admit all patients, ideally to a monitored bed.

48 Cardiomyopathy

Cardiomyopathies are diseases of the heart muscle associated with mechanical or electrical cardiac dysfunction, typically with ventricular hypertrophy, rigidity or dilatation. Cardiomyopathy is the major cause of heart failure in Africa, occurring at any age, but most commonly in the third and fourth decades of life, and mostly affecting men.

The first five minutes

- ABC, VS, facemask O_2, IV, cardiac monitor
- ECG

Classification

WHO define five broad categories, based on anatomy and physiology:

1. Dilated cardiomyopathy (DCM) – most common (80%) cardiomyopathy in Africa. Causes include untreated hypertension, infectious myocarditis, autoimmune diseases, alcohol, nutritional deficiencies, pregnancy.
2. Restrictive cardiomyopathy (RCM) – endomyocardial fibrosis (dense fibrosis of unknown aetiology).
3. Hypertrophic cardiomyopathy (HCM) – genetic cardiac hypertrophy.
4. Arrhythmogenic RV cardiomyopathy/dysplasia (ARVC/D) – dilation and reduction of systolic function of the RV

and loss of myocardial cells. Familial in about 50%.
5. Unclassified include fibroelastosis and mitochondrial disease.

History and physical examination

Key historical features

Cardiac risk factors, sudden death in the family, prior medical visits, cardiac surgery or congenital heart disease, hypertension, medication, history (chemotherapeutic agents, antiretroviral), toxin exposure (cocaine and ethanol).

Signs and symptoms

Heart failure: shortness of breath, chest pain, palpitations, syncope or near syncope, exercise intolerance, raised JVP, basal crepitations, peripheral oedema and S3 or S4 gallop.

Differential diagnosis

Acute coronary syndrome, myocarditis, pericarditis, periparitum cardiomyopathy, CCF.

Investigations

- Labs: CBC, electrolytes, renal, ABG ◇; cardiac enzymes ◈
- ECG: ◇ (LVH or other chamber enlargement, nonspecific ST-T wave changes, Q waves and AV conduction delay (characteristic of DCM); LBBB, deep narrow Q waves in lateral leads (characteristic of HCM))
- Imaging: CXR (enlarged cardiac silhouette, features of CCF) ◇; echo (diagnostic modality of choice; may show dilated chambers or muscle hypertrophy, decrease systolic or diastolic functions) ◈

Treatment

The goal of acute management is to reduce pre- and after-load to improve cardiac function and delivery of blood and oxygen to tissues.

Acute therapy

DCM

Treat for CCF (📖 p. 104)

HCM

Acute therapy is mostly supportive:
- O_2; target normal sats
- Maintain preload with IVF (if hypovolemic): initial bolus 250 ml (5 ml/kg in children). Repeat as needed (caution for overload)
- In CCF: avoid inotropes, careful use of diuretics, avoid digoxin and use vasodilators only if systolic dysfunction
- Control atrial fibrillation (📖 p. 123).

RCM

Acute therapy is mostly supportive:
- O_2; target normal sats
- Diuretics (cautiously) – IV furosemide ◇ (titrate to response)
- Control HR and maintain sinus rhythm

Critical documentation

Document cardiac rhythm, history of syncope, cardiac ultrasound, response to oxygen and initial therapy and

carefully record specific features necessitating intubation, if done.

Disposition

Admit all acutely unwell patients, preferably to ICU.

49 Myocarditis

Myocarditis is inflammation of the heart muscle leading to functional abnormalities. Clinical presentation may range from a flu-like illness to acute, severe cardiac failure progressing rapidly to shock and death.

The first five minutes

- ABC, VS, O_2, IV, pulse oximetry, cardiac monitor
- ECG, bedside cardiac US

Key historical features

- Constitutional symptoms, fever, fatigue, myalgia, chest pain
- Cardiac failure (dyspnoea, oedema), arrhythmias (palpitations, syncope)

Signs and symptoms

- Fever, tachycardia (often greater than expected), features of CCF, arrhythmias, hypotension, shock
- Children: as above, but signs of cardiac failure may be subtle – crying, hepatomegaly, respiratory distress, sweating, poor feeding, poor peripheral perfusion, tachycardia. Consider congenital heart disease

Possible causes and differential diagnosis

May be caused by infection, auto-immune disorders, drugs, toxins, envenomations. Most cases caused by viral infection (particularly enteroviruses and adenovirus).

Consider any other cause of cardiac failure, particularly IHD, arrhythmias, IE, rheumatic fever, pericarditis, sepsis etc.

Investigations

Initial lab testing is seldom useful. Cardiac enzymes are neither sensitive nor specific. Testing should be guided by suspected aetiology.

- Labs: CBC, electrolytes, renal, ESR/CRP ◇
- ECG: ◇ (sinus tachycardia, tachy- and brady-arrhythmias, conduction abnormalities, non-specific QRS, ST and T-wave abnormalities)
- Imaging: CXR ◇ (heart may be normal or enlarged, features of CCF); echo ◈ (may provide useful information)

Management

The goal of acute management is maintenance of cardiac output, oxygenation and perfusion.

No specific treatment exists, though IV immunoglobulin ◈ treatment for paediatric viral myocarditis is associated with improved outcomes. Treatment is supportive (treat CCF and arrhythmias) with consideration for transplant in selected critically ill patients.

Disposition

Admit all patients with myocarditis.

50 Approach to atrial fibrillation (AF)

In AF, atria contract rapidly with variable transmission to ventricles, resulting in the ‘irregularly irregular’ pulse and

ECG pattern. Most often associated with structural disease of atria (valve disease, congenital, hypertensive, cardiomyopathy) or transient atrial 'stretch' from volume overload (post-operative fluid shifts). AF may occur spontaneously in young patients with no structural disease ('lone' a-fib), or be permanent or paroxysmal. Atrial remodelling occurs; recurrent episodes increase risk of persistent AF. AF is triggered by stressors that can cause sinus tachycardia (e.g. infection, dehydration, hyperthyroid withdrawal, PE, MI) so always evaluate for cause.

The first five minutes

ABC, VS, O_2, IV, monitor. Address causes of shock (e.g. dehydration, sepsis, MI). Prepare for cardioversion (📖 p. 90 and p. 93) if shock, ischaemia, pulmonary oedema, or AMS with no other cause and ventricular rate ≥140

History and physical

Medications/drugs (theophylline, digitalis, caffeine, alcohol). Asymptomatic or with palpitations, DIB, fatigue, dizziness or syncope. Irregularly irregular pulse.

Differential diagnosis

Sinus arrhythmia, atrial flutter, Mobitz block, ventricular tachycardia.

Investigations

ECG and follow-up echocardiogram ◇. Other tests per suspected aetiology.

Management

The goal of acute management is restoration of perfusion, identification of triggers, and referral for chronic management. Short term risk of embolic complications (e.g. stroke) is low; anti-coagulation can be inpatient.

- Unstable or with rapid ventricular response (RVR)
- Treating triggers may resolve shock and RVR
- Unstable: immediate synchronised cardioversion (📖 p. 93) ◈
- Rate control: caution as cardiac output may depend on rate. Consider allowing rates of < 130s acutely if no ischaemia. Rate < 140 unlikely to cause ↓BP
- Agents: nodal blockade with diltiazem or BB (↓BP may limit dosing, avoid using CCB and BB together); digoxin (less hypotension, slow onset, dangerous toxicity); amiodorone (slow onset, less hypotension)
- Stable with normal rate: chronic management may include rate control, anticoagulation, rhythm control or conversion, and management of underlying conditions. Some sites will cardiovert stable AF of <48h duration in the acute setting, but this requires a local protocol. Never cardiovert a stable patient who has been in AF > 48 hours as atrial clot may embolise. These patients require echo and anti-coagulation prior to conversion

Disposition

Admit all patients with new AF. Admit haemodynamic instability to ICU. ◈

51 Pericarditis

Pericarditis is inflammation, infection or infiltration of the pericardial sac. The outer layer of the myocardium is often also involved. Many cases are idiopathic, but common causes include infection (viral, TB), uraemia, auto-immune inflammation (SLE), post infarction (Dressler syndrome), malignant infiltration and drug reactions.

Pericarditis may cause pericardial effusions (rapidly forming effusions may lead to tamponade). Accompanying myocardial inflammation may lead to arrhythmias and conduction abnormalities.

The first five minutes

- ABC, VS, O_2, IV, pulse oximeter, cardiac monitor
- Bedside cardiac US

• ECG

History and physical examination

The cardinal features of pericarditis are chest pain and tachycardia.

Key historical features

Viral prodrome; constitutional symptoms and other features of TB; conditions that may lead to pericarditis (renal failure, malignancy, drug use, trauma).

Signs and symptoms

Chest pain (central, burning, constant, often positional), palpitations, symptoms of cardiac failure or tamponade (dyspnoea, oedema). Features of systemic infection or TB may be present. Search for arrhythmias and signs of cardiac failure. A pericardial friction rub may be heard at the lower left sternal border with patient sitting up, leaning forward and in full expiration. Search for features of early tamponade.

Differential diagnosis

Any cause of chest pain (ACS, PE, AD, coronary artery spasm, oesophageal reflux, myocarditis).

Investigations

- ECG ◇: diffuse concave up ST elevation – usually not in V1 or aVR – and PR segment depression. PR elevation in aVR; as inflammation resolves ST and PR normalisation, followed by T-wave inversion is common
- Imaging: CXR ◇ (often normal; may reveal features of CCF or an enlarged cardiac silhouette); echo ◈ (if effusion present, look for tamponade: RA and RV collapse and septum bowing)
- Pericardial fluid aspirate ◈ (test fluid for cell count, Gram stain and bacterial culture, acid fast smear/culture, cytology, protein, LDH and adenosine deaminase concentrations; TB pericarditis: acid fast bacilli by smear or culture (50% of cases) and high protein and leukocyte count with lymphocyte and monocyte predominance)

Management

The goal of acute management is restoration of perfusion.

Consider emergency pericardiocentesis if any evidence of tamponade. Further management should be directed at symptoms (analgaesia), complications (arrhythmias, CCF) and ultimately finding and reversing the cause.

- Uncomplicated pericarditis:
  - » Anti-inflammatory medications ◇: indomethacin 25–75 mg Q6h or ibuprofen 600 mg × 7 days Q6h
  - » If recurrent or refractory, consider corticosteroids
- TB pericarditis:
  - » Start the patient on anti-TB drugs as per local guidelines
  - » Prednisolone (evidence mixed): 60 mg daily tapering dose by 10 mg each week for four weeks total
  - » In constrictive pericarditis consult surgery for pericardiectomy
- Other:
  - » Bacterial: aggressive IV antibiotics and early surgical consult
  - » Uraemic: dialysis
  - » Dressler (post-MI): cardiology referral

Disposition

Admit patients with acute effusion, arrhythmias, fever, and those requiring periocardiocentesis, or with abnormal VS.

52 Congenital heart disease

Congenital heart disease is estimated to have an incidence of 8:1 000. Early presentation frequently mimics other,

more common illnesses, such as pneumonia or septicaemia, causing a delay in diagnosis and leading to complications. It is crucial that children with CHD are detected as soon as possible; ideally at their first point of contact with health care.

The first five minutes

- ABC, O_2 sat (both hands)
- Bedside cardiac US

Categories of CHD

Note that the exact diagnosis of the underlying lesion is usually not required to provide life supporting management. Acute presentation is one of a combination of: cyanosis, cardiac failure, shock.

Acyanotic heart disease

- Intracardiac left to right shunts (e.g. VSD, ASD)
- Obstructive lesions (e.g. critical pumonary stenosis, aortic stenosis, coarctation, interrupted aortic arch)

Clinical features

- Murmur, failure to thrive, cardiomegaly, cardiac failure, recurrent pneumonia
- Cardiogenic shock: poor perfusion and poor femoral pulses may indicate critical stenosis in the aorta at some level (e.g. valvular, interruption, coarctation) or critical pulmonary stenosis in a neonate

Cyanotic heart disease

- RV outflow tract obstructions (e.g. tetralogy of Fallot, pulmonary atresia with VSD)
- Common mixers (e.g. truncus arteriosus, TAPVD)
- Common mixers with pulmonary outflow obstruction (e.g. tricuspid atresia, double outflow right ventricle with pulmonary stenosis)
- Parallel circulations (e.g. transposition of the great arteries)

Clinical features

Percutaneous saturations and hyperoxia test:

- With the patient breathing room air, ABG, give 100% O_2 for 10 minutes; repeat ABG – if pO_2 still < 100 mmHg (13.0 kPa), diagnosis of cyanotic CHD is highly likely
- Differential sats in a neonate (compare SaO_2 between the right thumb (preductal) and a toe – any patient with a SaO_2 less than 97% or a difference of more than 2% should be referred to a cardiologist)
- Children with mild cyanotic CHD generally look well, without respiratory distress. A CXR helps to assess lung perfusion (dark lung fields); murmurs may be nonspecific or absent

Management

The goal of acute management is support of perfusion, confirmation of diagnosis, and initiation of specific therapy. Expert help should be involved early.

Acyanotic

When in cardiac failure:

- Diuresis ◇: furosemide 1 mg/kg IV
- Ventilation ◈: NIPPV (e.g. CPAP) ameliorates pulmonary oedema
- If shocked or persistently hypotensive, consider dopamine ± milrinone infusion ◈ OR dobutamine ◈

Cyanotic lesions

- Treat all neonatal cyanotic CHD as ductal dependent until proven otherwise:
  - Prostaglandin E_1 ◈ 0.05–0.1 mcg/kg/min, titrate to O_2 improvement. Causes hypotension and tachycardia; bolus IVF accordingly (e.g. RL 5–10 ml/kg IV). Beware apnoea)
  - O_2, ventilation
  - Morphine ◇ for sedation
  - Urgent referral.
- Hypercyanotic spell:
  - A spell may be the initial presentation of a child with tetralogy of Fallot *at any age*. Other cyanotic lesions such as tricuspid atresia or double outlet right ventricle with pulmonary stenosis can also spell. Treatment includes fluid resuscitation, effective sedation, correction of acidosis and early referral
- Obstructive lesions: Prostaglandin E_2 ◈ as above to maintain perfusion of the lower body by a right to left shunt through the patent duct

Disposition

Refer all confirmed or suspected CHD patients to a paediatric cardiology centre.

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Part

4

B. Dental and oral surgery

53 Approach to dental pain

While generally benign, dental pain may be associated with airway compromise, aspiration, septicaemia, and cerebral spread of infection.

The first five minutes

- ABC (ensure no foreign body (including teeth) in oro-pharynx), VS, consider C-spine immobilisation as needed
- Evaluate for pain or difficulty with swallowing

History and physical examination

Key historical features

Trismus, pain with biting, fever, respiratory complaints, bleeding gums, headache, neurological symptoms. Recent trauma or dental procedures. Pain medication and antibiotic use, immunocompromise, coagulopathy, cardiac valvular disease (may need antibiotics for procedures).

Signs and symptoms

- ABC: stridor, swelling of soft tissues of mouth, elevated tongue
- VS: fever, tachypnoea, tachycardia
- Face/neck: tenderness, oedema, erythema or other signs of cellulitis
- Oro-pharynx: check soft tissues, gums, tongue for swelling, laceration, ulcers, bleeding. Examine teeth integrity and alignment. Identify which teeth are involved. Palpate teeth and examine maxilla/mandible for stability and tenderness. Percuss with tongue blade to identify offending tooth by tone/tenderness

Possible causes and management

The goal of acute management is airway protection, identification and treatment of serious conditions, and analgesia. General management of dental pain includes NSAIDs and paracetamol; opiates and dental blocks for refractory pain or procedures (📖 Dental blocks p. 809).

Traumatic

- Dental fractures (📖 p. 138)
- Tooth subluxation: loose but not displaced. Requires splinting only
- Tooth luxation: loose and displaced. Lateral: reposition and splint. Intrusion (pushed toward alveolar bone): no immediate treatment required. Extrusion (partial avulsion): reposition and splint

- Tooth avulsion: displaced from oral cavity. Handle tooth by crown only. Immediately place in most physiologic solution available (best to worst – Hank's Balanced Salt Solution, milk, saliva (may transport in patient's or parent's mouth)). Irrigate socket, achieve haemostasis, replace tooth, splint and refer to dentist. Do not re-implant primary teeth
- Soft tissue injury/intraoral haemorrhage: ensure haemostasis with gauze/direct pressure. Suture lacerations with 4–0 absorbable sutures

Non-traumatic

- Dental caries ± periapical abscess: sensitive to cold, tender to percussion, necrotic tooth, swollen at base. May require filling, extraction, incision and drainage of abscess
- Gingivitis/periodontitis: inflamed gums, bleed with brushing (commonly pregnant or DM) (📖 p. 135)
- Acute necrotising ulcerative gingivitis (ANUG): halitosis, fever, malaise; triad of pain, ulcerated papillae, gingival bleeding. Occurs with immunocompromise, measles, malaria, intestinal parasites.
- Cracked tooth/split root: pain with chewing (or biting tongue blade), often traumatic or secondary to dental procedure. Analgesics, dental referral
- Alveolar osteitis (dry socket): pain with clot displacement 2–3 days after tooth extraction. Irrigation, packing with iodoform gauze, analgesics, antibiotics, daily follow-up
- Periocoronitis: pain with tooth eruption of 3rd molar. Refer dentist
- Ludwig's angina: deep space infection with swelling of submandibular, submental, and sublingual spaces with elevation of tongue and bilateral brawny/woody induration. Evaluate for fever, difficulty swallowing. This is a life-threatening emergency; early and aggressive airway protection is essential
- Other: exclude referred pain of trigeminal neuralgia

Investigations

- Labs: as indicated for systemic illness or procedures
- Imaging: XR mandible/face (visualise dental crown, apices, surrounding bone), Panorex (best visualisation of dental apices, maxilla, mandible), CXR (if missing whole or partial tooth to exclude aspiration) ◇. Contrast CT face (non-traumatic – evaluate for deep space infection) ◈

Critical documentation

Document airway status, location of teeth/fragments, signs of systemic infection, evaluation for head injury if traumatic, stability of other teeth and intact alveolar plate.

Disposition

Discharge most patients to dentist. Admit systemically ill patients with Ludwig's angina and those who may require prolonged mechanical ventilation ◈.

54 Approach to lesions of the oral cavity

Oral lesions are often the first clues to the diagnosis of systemic diseases. Recognising oral abnormalities helps direct investigations and subsequent management.

Abnormalities of the tongue

Benign lesions

- Geographic tongue: map-like appearance due to irregular patches on tongue surface. Management: antihistamine gel or steroid mouthwash
- Black hairy tongue: lengthening of papillae caused by excess bacteria and overgrowth of yeast. Management: improve oral hygiene, brush tongue, quit smoking

Systemic disease

In any of these conditions, management centres on evaluation and treatment of underlying disease.

- Strawberry tongue: an inflamed bright red tongue caused by *Streptococcus pyogenes*, seen in Kawasaki disease, STSS, scarlet fever. Use oral antibiotics
- Macroglossia: enlarged tongue seen in acromegaly, amyloidosis, hypothyrodism, and mucopolysacharidosis
- Microstomia: systemic sclerosis
- Xerostomia: mouth breathing, dehydration, cystic fibrosis, Sjögren syndrome, sarcoidosis, DM, anticholinergics. Use saliva substitutes, saliva stimulants
- Atrophic tongue: unilateral – CN 12 palsy; bilateral – motor neuron disease

Precancer and cancer

- Leukoplakia: most common oral precancer, (white patch or plaque that cannot be scraped off). Management: biopsy, removal if dysplastic
- Oral cancer: most common squamous cell carcinoma. Predisposing factor is chewing tobacco (toombak in Sudan). Management: related to site and stage of disease

Abnormalities of the mucosa

Benign lesions

- Torus mandibularis and palatines: bony growths on floor of mouth and hard palate, usually asymptomatic. Management: no treatment needed; if symptomatic, surgical removal

Systemic disease

In any of these conditions, management centres on evaluation and treatment of underlying disease.

- Koplik's spots: prodromal stage of measles (see image section)
- Purpuric spots: thrombocytopenia, rheumatoid arthritis, acute leukaemia or aplastic anaemia
- Ulcerations:
  - » Aphthous: shallow painful, erythematous ulcers; seen with HIV, collagen disorders, Crohn's, Behcet's syndrome, drugs (NSAIDs, ACE inhibitors)
  - » Traumatic: from direct trauma (ill-fitting dentures, burns, restorations). Management: remove sources
  - » Ulcers and bullae: caused by pemphigus vulgaris and bullous pemphigoid, drug-induced, and Stevens-Johnson syndrome

Pre-cancer and cancer

- Lichen planus: trabecular pattern on inner surfaces of the cheeks; associated with ACE inhibitors, beta-blockers. Management: topical steroids, consider discontinuation of medications
- Burkitt's lymphoma: malignant B-lymphocyte lymphoma, common in Africa; often presents with hard swelling that causes bone destruction, tooth loss, facial deformity. Manage underlying disease
- Leukaemia: oral candidiasis, herpetic infections, gingival and soft plate petechial haemorrhages, ulcers. Manage underlying disease

Abnormalities of the gingiva

- Gingival hyperplasia: pregnancy, phenytoin, cyclosporine and calcium channel blockers (nifedipine) and acute leukaemia. Management: oral hygiene, discontinuation of medications, gingivectomy
- Pyogenic granuloma: benign proliferation of gingiva due to local trauma or irritation. Management: only if symptomatic; surgery, electrocautery

Infections of the oral cavity

- Oral candidiasis (thrush): predisposing factors: immunocompromised, antibiotics, inhaled steroids. Management:

topical oral antifungal, evaluate for and treat underlying disease
- Herpes simplex (type 1 and 2): acute recurrent painful vesicular eruptions which ulcerate, followed by secondary infection. Management: acyclovir, analgesics
- Herpes zoster: severe pain followed by unilateral vesicular eruptions along dermatome of the trigeminal nerve presenting as tooth pain. Management: acyclovir, analgesics
- Herpangina (coxsackie, echoviruses): shallow painful ulcers in soft palate, uvula, posterior pharynx and tonsillar pillars; sparing tongue, buccocal mucosa and gingiva. Management: analgesics
- Hand-foot-and-mouth disease: painful shallow ulcers with a red halo on the tongue, gingiva, soft palate, and buccal mucosa. *Other parts:* buttocks, palms and plantars. Management: analgesics
- Gonorrhoea: pharyngitis involving the uvula and tonsils and may present with or without pustules or exudates. Management: antibiotics, analgesics (p. 966)
- Human papillomavirus (HPV): oral lesions similar to condyloma acuminatum, or venereal warts. Management: evaluate for and treat underlying disease
- Syphilis: primary chancre: lip, tongue or tonsils. Secondary: multiple, oval-shaped, slightly raised ulcers or erosions covered with a gray membrane tongue and condyloma lata. Congenital perforation of the palate. Management: antibiotics, analgesics, treat underlying disease
- HIV: *Early:* sore throat, mucosal erythema, focal ulceration. *Late:* Pseudomembranous candidiasis, linear gingival erythema, herpes simplex, hairy leukoplakia, Kaposi sarcoma, lymphoma. Management (p. 130)

Oral manifestations of systemic diseases

In any of these conditions, management centres on evaluation and treatment of underlying disease.

- Pigmentation (lips and buccal mucosa): Peutz-Jeghers syndrome, Addison's disease, haemosiderosis, multiple pregnancies, lead poisoning, and drugs
- Diabetes mellitus: gingival and periodontal inflammation, spontaneously bleeding gums, halitosis, predisposition to fungal infection
- Gigantism and agromegaly: coarse facial appearance, prognathism, and spacing of the teeth
- Hypothyroidism: lip thickening, failing of teeth eruption
- Marfan's syndrome: high arched palate, crowded mal-occluded teeth
- Ehlers-Danlos syndrome: hypoplasia of the enamel, periodontitis; fragile, easily tearing mucosa and gingival
- Crohn's disease: swollen lips, chelosis, fissuring of the buccal mucosa
- Uremic stomatitis: painful oral disorder with white plaques or crusts
- Bulimia nervosa: self-induced vomiting leading to lingual dental surfaces erosions
- Vitamin deficiencies and iron deficiency anaemia: angular chelitis, dryness, glossitis with painful and burning tongue, aphthous-like ulcers and atrophy of oral mucosa
- Vitamin C deficiency: 'scurvy', spares the tongue, gingivitis with spontaneous haemorrhage, ulceration, tooth mobility and periodontitis (scorbutic gingivitis)
- Zinc deficiency: periorofacial and acral psoriasiform erythematous rash

55 Dental and other oral infections

While often benign, dental and other oral infections may cause airway compromise, aspiration, septicaemia, osteomyelitis, cerebral infections, and infections in the superficial and deep spaces of the neck.

The first five minutes

- ABC, VS

History and physical examination

Key historical features

Location and onset of symptoms, history of trauma, dental extraction or other oral surgery, difficulty or pain on swallowing or mouth opening. Ask about systemic symptoms (dyspnoea, chest pain, neck pain, fevers/chills), and

immunocompromise (HIV, DM).

Signs and symptoms

Assess the airway, look for oedema, pus, caries, swollen mouth floor. Check for trismus, meningismus, and lymphadenopathy.

Possible causes and management

Dentoalveolar infections

- Dental caries: cavities from plaque and decay. Analgesics and dental referral
- Pulpitis: inflammation of pulp; severe toothache, worse with cold or recumbence. Analgesics and referral
- Periapical abscess: infection of alveolar bone. Antibiotics; analgesia; referral

Periodontal disease

- Gingivitis: inflammation of the gums causing swelling, easy bleeding, possibly halitosis. Improved oral hygiene, Chlorhexidine 0.12% mouthwash, NSAIDs
- Acute necrotising ulcerative gingivitis: infection of the gums ('trench mouth'); pain, grey pseudo-membranes, altered taste, fever, malaise, LAN. Oral hygiene, Chlorhexidine 0.12% mouthwash, NSAIDs, oral antibiotics
- Periodontitis: inflammation and progressive loss of tissues connecting teeth to bone. Oral hygiene, mouthwash, oral antibiotics, referral
- Periodontal abscess: acute infection of supporting structures, often seen in diabetics. Causes red, fluctuant area of gingiva with expression of pus. I&D, oral antibiotics, referral
- Pericoronitis: local infection caused by food and micro-organisms trapped under gum flaps during eruption of permanent/wisdom teeth, causing painful mastication and swallowing, facial swelling, halitosis, LAN. Oral hygiene, irrigation, PO antibiotics, referral for extraction

Dentoalveolar and periodonatal oral antibiotic choices:

- Empiric antibiotic regimens: (amoxacillin 500 mg TID OR (penicillin 500 mg Q6h AND metronidazole 400 mg tds)) OR (amoxacillin – clavulanic acid 875 – 1 g PO bd) OR (clindamycin 300 mg tds) OR (doxycycline 100 mg bd). All for 10 days

Superficial space infections

Buccal (cheek swelling), submental (chin swelling/firmness), masticator (trismus and pain near mandibular body/ramus, mimics osteomyelitis), canine (upper lip/periorbital swelling, may be complicated by purulent maxillary sinusitis), infratemporal (trismus and pain).

- All need I&D and/or antibiotics
- Superficial space infection IV antibiotic: 1st line: ampicillin-sulbactam; 2nd line: either (penicillin AND metronidazole) OR clindamycin 2–6 weeks

Deep space infections

Secure airway, IV antibiotics, and emergency surgery/ENT consultation.

- Submandibular (Ludwig's angina): bilateral infection beginning in mouth floor; can result in airway obstruction and mediastinitis
- Parapharyngeal (Lemierre's disease or suppurative jugular thrombophlebitis): of space containing carotid sheath and CN IX–XII
- Retropharyngeal abscess: infection behind pharynx, commonly occurring in children
- 'Danger space': space extending from skull base through posterior mediastinum, predisposes to mediastinitis and intracranial spread
- Prevertebral: space extending from skull base to coccyx; difficult diagnosis
- Peritonsillar (peritonsillar abscess): infection around tonsil. Suspect in patients with unilateral sore throat, uvular deviation, soft palate fullness, muffled voice, fever. Attempt careful I&D (see 🕮 Peritonsillar abscess aspiration,

p. 830)
• Parotid (acute suppurative parotitis): suspect in patients with sudden unilateral induration of cheek and purulence from parotid duct

Deep space infection IV antibiotic choices: (📖 p. 968)

Investigations

• Labs: Gram stain and culture (to determine sensitivities) ◇
• Imaging: US (useful but may not rule out extension) ◇; CT (preferred) ◈

Critical documentation

Airway involvement, crepitus, intra-oral vs. extra-oral findings, immunocompromise.

Disposition

Admit patients with systemic illness or with risk factors. Discharge and follow up most other patients.

56 Dental fractures

This chapter deals with injuries to the teeth; see 📖 p. 761 for other facial fractures.

The first five minutes

• ABC (ensure no foreign body (including teeth) in oro-pharynx), VS, consider C-spine immobilisation

Tooth anatomy

• *Enamel:* outermost layer, covers the crown (the part of the tooth visible above the gum line)
• *Dentine:* middle and largest layer of the tooth, is porous
• *Pulp:* innermost layer; contains sensitive neurovascular supply
• *Root:* part of the tooth beneath the gum line; composed two layers (dentin and pulp) and covered with a thin layer of cementum

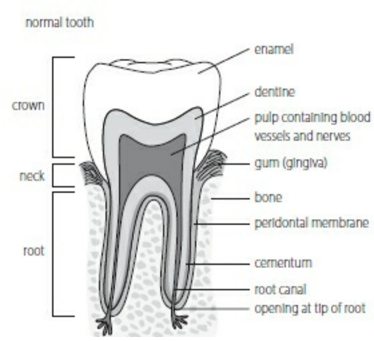

Figure 56.1 Anatomy of the tooth

Classification

Uncomplicated fractures through the enamel

Ellis I – not sensitive to temperature or percussion. Risk of pulp necrosis < 1%.

Uncomplicated fractures through the enamel and dentin

Ellis II – yellow dentine identified in contrast to white enamel surface. Sensitive to temperature and percussion. Risk

of pulp necrosis 10%; continues to increase over hours until treated. Risk of necrosis higher in age < 12 years (higher pulp to dentin ratio).

Complicated fractures of the crown involving the pulp

Ellis III – extend through all three layers. Pulp identified by pink hue. Often painful, may be painless if innervation has been disrupted. Even with optimal treatment, risk of pulp necrosis 30%; increases over hours until treated.

Management

The goal of acute management is to prevent infection and pulp necrosis which can lead to loss of the tooth or systemic complications.

Management is dictated by fracture type; careful examination and classification is essential.

- Identify associated injuries (including facial, head or cervical spine)
- Identify all fracture fragments, which may be swallowed, aspirated, embedded in intraoral lacerations, or fully displaced into the alveolar bone and surrounding structures. CXR to assist in locating all teeth and fragments
- Tetanus as needed
- Antibiotic coverage (e.g. penicillin V 250 mg qid 5–10 days) for any fracture exposing dentin or pulp
- Discharge instructions should include a soft diet and infection warning signs

Ellis I: conservative management: smooth or file sharp edges to prevent injury to oral soft tissues.

Ellis II: cover with a dressing such as calcium hydroxide ◇, zinc oxide, or glass ionomer composites ◇.

Ellis III: refer to dentist or oral surgeon for urgent pulpectomy. If not immediately available, address pain (📖 p. 52) and cover fracture with a dressing (as above). Brisk bleeding from the pulp can interfere with dressing application; inject a small amount of local anaesthetic with adrenaline. ◇

Critical documentation

Document exact depth of fracture, location of teeth/fragments, airway status, evaluation for head injury, stability of other teeth and intact alveolar plate.

Disposition

Discharge patients with isolated dental fractures to the care of a dentist.

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Part

4

C. Dermatology

57 Describing skin lesions

There are more than 3 000 different dermatological diagnoses. The ability to describe what you see will aid diagnosis, management, and consultation.

Equipment

- Measuring tape or ruler
- Magnifying glass
- Body map (a burns chart will work)

Getting advice

A picture paints a thousand words. Photos sent by SMS or email aid in remote access diagnosis (but always document the patient's consent and consider local policies regarding clinical images).

Terminology

General

Lesion: single area of abnormal skin
Rash: group of lesions
Confluent lesions: smaller lesions that merge

Layer of skin affected

Epidermis: outer layer of the skin
Dermis: connective tissue layer under the epidermis, contains nerve endings and blood vessels
Subcutaneous tissue: adipose (fat) layer under the dermis, contains nerves and blood vessels

Distribution

Acral: affects hands, feet, ears or nose

Dermatomal: follows nerve root distribution
Follicular: arising from hair follicles
Generalised: wider spread (describe as mild, severe, scattered or diffuse)
Herpetiform: clusters of vesicles
Photosensitive: affecting sun-exposed areas only
Exanthema: widespread rash associated with flu-like symptoms

Pattern

Nummular/discoid: coin shaped
Linear: in lines
Target: dark/crusted centre and outer ring around a pale, raised middle ring
Annular: lesions arranged in a circle
Morbilliform: lacy, generalised smooth pink-to-red macules or papules that blanch

Colour

Hyperpigmentation: darker skin than surrounding area
Hypopigmentation: paler skin than surrounding
Erythema: red skin that blanches on pressure
Erythroderma: erythema which affects the whole (or nearly the whole) body

Morphology

Macule: smooth area of colour change ≤1.5 cm diameter
Patch: larger smooth area of colour change
Papule: small, palpable lesions <0.5 cm (some authors allow ≤ 1.5 cm diameter) (solitary or multiple)
Nodule: solid, larger papule
Cyst: papule or nodule that contains fluid
Plaque: palpable, flat lesion ≥ 0.5 cm diameter (can be raised or level with skin)
Petechiae: purpuric lesions ≤ 3 mm (red, purple or brown)
Purpura: haemorrhage of small skin vessels with no blanching on pressure
Telangiectasia: prominent skin vessels
Vesicle: fluid filled blister ≤ 0.5 cm diameter (solitary or multiple)
Pustule: vesicle containing pus
Bulla: larger fluid filled blister (single or multi-loculated, solitary or multiple)
Abscess: localised collection of pus
Wheal: oedematous papule/plaque due to dermal swelling (indicates urticaria/hives)

Surface

Smooth
Hyperkeratosis: scaling (appears to have transparent, superficial thin layers), lichenoid (adhered to skin), keratotic (horny), exfoliating (peeling), macerating (moist peeling), verrucous (warty)

Secondary changes

Lichenification: thickened skin
Crusting: dried exudate or blood
Excoriation: scratch marks
Erosion: skin surface loss (usually shallow and moist, or crusted)
Fissure: thin crack in skin (usually as a result of dryness)
Fungating: fungus or mushroom-like growth
Granulation: seen in normal wound healing and consists of clumps of new capillaries and fibrous tissue
Ulcer: full thickness epidermal loss. When crusted the crust is refer to as an eschar
Hypertrophy: excessive skin growth usually associated with scars

Atrophy: thinned skin

58 Approach to skin rash

Skin conditions account for up to 10% of emergency visits. Significant systemic illness may initially present as a rash, or the rash may be an advanced stage of a severe disease. Most rashes do not require emergency intervention. Life threatening rashes often have a sudden onset, are widespread, involve mucous membranes or present as blisters/purpura.

The first five minutes

ABC, VS, O2; IVF as needed.

Rashes associated with fever, tachycardia or hypotension, or in an otherwise ill-appearing patient may indicate life threatening causes.

History and physical examination

Key historical features

Onset, duration, rate of progression, location and distribution, fever, pain, itching, discharge. Recent/current illness, drugs, travel or contacts. Exposure to plants, chemicals or new creams, soaps or other household products. Pre-existing illness, immunocompromise. Family and sexual history.

Signs and symptoms

Examine an undressed patient under adequate lighting; include hair, nails and mucosal surfaces. Note the number, size and colour of lesions, body regions involved, symmetry, shape, arrangement, scaling, crusted, palpable lesions (📖 p. 142). Excoriations represent pruritis even if not reported. Nikolsky sign (blister formation or separation of epidermis on light rubbing of unaffected skin) is a sign of toxic epidermal necrolysis (TEN) (📖 p. 165), staphylococcal scalded skin syndrome (SSSS) and pemphigus vulgaris bullous (📖 p. 150) (it does not occur in bullous pemphigoid).

Examination may be facilitated by a dermoscope (for scabies, pigmented lesions to diagnose melanoma, etc.) or Wood's lamp (to diagnose tinea and other infections).

Urticarial lesions or 'hives' (often intensely itchy and associated with dermatographia – immediate appearance of raised lesions with superficial scratching of skin) usually represent an allergic process, but may be associated with a range of conditions. Screen all patients with hives for anaphylaxis (📖 p. 38).

Investigations

Most diagnoses are clinical; adjuncts can be utilised as needed and as directed by the suspected diagnosis. Include skin swabs, smears, biopsy and scrapings for microscopy, culture, and cytology as needed.

Management

Management generally consists of symptomatic treatment (see below), identification of the cause, and treating the cause (📖 Dermatology therapy p. 984).

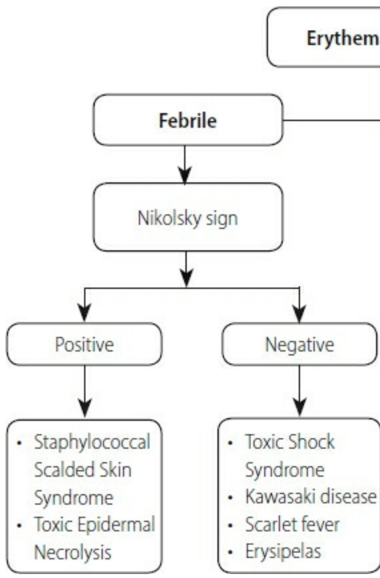
- Moisturisers: aqueous cream (not petroleum jelly), urea ointment/cream
- Ointments (chronic, dry lesions) and creams (acute, wet lesions)
- Coal tar paste/ointment: anti-inflammatory, anti-itch (causes photosensitivity – use at night only)
- Salicylic acid ointment: dissolves scales, softens thickened skin
- Potassium permanganate solution: decreases oozing, antiseptic, antifungal (mild)
- Gentian violet paint: antifungal, antiseptic (stains skin)
- Sulphur: antiseptic (dries skin)
- Irritant/allergen avoidance
- Hygiene

• Specific treatments (see other chapters)

Disposition

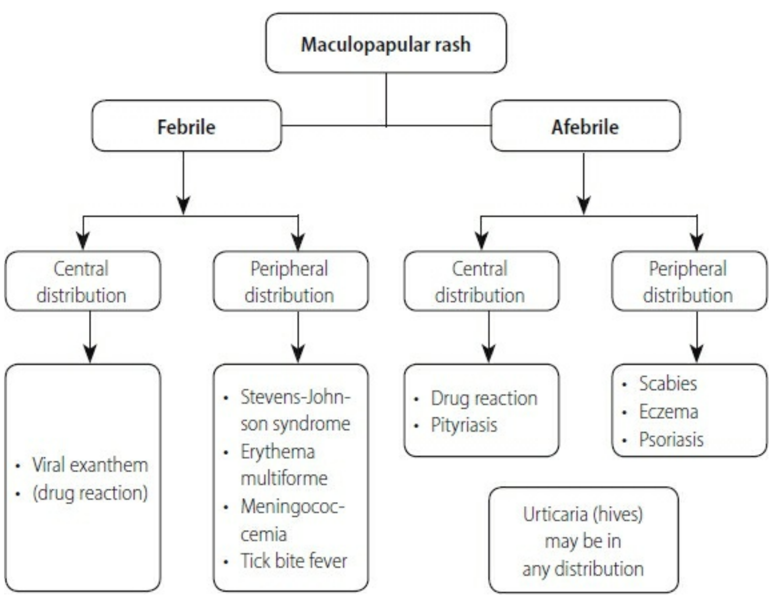
Admit acutely unwell patients. Most cases can be followed up in a primary care setting. Consider dermatology referral if unsure. Involve a specialist early if you suspect malignancy.

Figure 58.1 Algorithmic approach to diagnosing the unknown erythematous rash



Adapted from Murphy-Lavoie H. Emergent diagnosis of the unknown rash, the algorithmic approach. *Emergency medicine* 2010:42(3):6-17.

Figure 58.2 Algorithmic approach to diagnosing the unknown maculopapular rash



Adapted from Murphy-Lavoie H. Emergent diagnosis of the unknown rash, the algorithmic approach. *Emergency medicine* 2010:42(3):6.

59 Approach to the child with a rash

Conditions associated with rash in a child range from benign to life-threatening. Most rashes do not require

emergency intervention, but it is important that the degree of risk be stratified early on. Life threatening rashes often have a sudden onset, are widespread, involve mucous membranes or present as blisters/purpura.

The first five minutes

ABC, VS, O_2 sat; IVF as needed.

Rashes associated with fever, tachycardia or hypotension may indicate life-threatening causes.

History and physical examination

Key historical features

Duration, rate of progression, location, pain, itching. Recent/current illness, medications, vaccinations, travel or contacts. New creams, soaps or other household products. Chronic illness, immunocompromise.

Signs and symptoms

Eczematous

Acute (redness, swelling, papules, blisters, oozing crusts) or chronic (lichenification, excoriations, scaling) form; infants (cheeks, trunk, extensor surfaces of extremities, knees and elbows) or children (neck, hands, feet, antecubital and popliteal fossa).

Viral exanthem

Measles, rubella, roseola, erythema infectiosum, EBV, CMV (macular/maculopapular); varicella/zoster, eczema herpeticum, coxsackie (vesicular and pustular); papular acrodermatitis of childhood, molluscum contagiosum (papular), pityriasis rosea (fine scaling, pine tree pattern).

Bacterial

Impetigo; cellulitis, lymphangitis; yaws (friable raspberry-like papule or nodule); scarlet fever (generalised, tiny, pinkish-red spots following sore throat/impetigo).

Parasitic

Schistosomiasis, trypanosomiasis, onchocerciasis (generalised pruritis); dracunculiasis (painful ulcerating lesion).

Mycobacterial

Leprosy (hypopigmented/erythematous macules, insensitive to touch, enlarged nerves); buruli ulcer (papule or nodule which ulcerates widely).

Fungal

Tinea capitis (scalp scaling, black dot tinea, inflammation or boggy tender kerion, papules and pustules, reactive adenopathy); tinea versicolor (macules and patches with fine scales, tan reddish or white); tinea corporis (annular plaque, raised advancing border, central clearing).

Drug eruptions

Variable appearance (📖 p. 165).

50% maculopapular, usually within two weeks of medication. Usually symmetric, often confluent.

Miscellaneous

Nappy/diaper rash (irritant contact rash from chafing in nappy area); pustular melanosis (present at birth, 2–4 mm transient vesicular-pustules, brown base after rupture); erythema toxicum (first week of life, 1–3 mm yellow-white

papules/ pustules on erythematous base).

Differential diagnosis

	Unwell	Well, mild illness	No illness
Erythematous	Urticaria, scarlet fever, staphylo-coccal scalded skin syndrome, Steven's-Johnson, toxic shock syndrome, rheumatic fever, SLE, sarcoidosis	Erythema multiforme, roseola, erythema infectiosum	Psoriasis, pityriasis rosea, nappy rash
Maculopapular	Meningococcaemia, endo-carditis, drug hypersensitivity, Kawasaki, measles, HIV sero-conversion, acute hepatitis B or C	Drug reaction, CMV, EBV, rubella	Tinea, scabies, impetigo, fol-liculitis
Vesicular-bullous	Steven's-Johnson, toxic epider-mal necrolysis	Chickenpox, coxsackie, congenital syphilis, gonorrhoea	Pustular mela-nosis, erythema toxicum
Purpuric	Meningococcaemia, leukaemia, Henoch-Schönlein purpura	Idiopathic thrombocyto-paenia, child abuse	Child abuse

Investigations

Most diagnoses are clinical. Adjuncts can be utilised as needed and as directed by the suspected diagnosis. Include skin swabs, smears, biopsy and scrapings as needed.

Management

Parasites or drug eruptions (📖 p. 162 and p. 165).

Eczematous

Topical corticosteroids – 1% hydrocortisone (face and nappy), mid to high potency (other areas); emollients following bath; avoid vaseline, mineral oils, fragrances. For itching use oral antihistamines. Treat supra-infection with oral antibiotic to cover *Staphylococcus aureus* (e.g. cloxacillin) and topical antimicrobial shampoo as soap (if weeping, bathe in potassium permanganate 1:4 000 solution).

Viral exanthema

Treat symptomatically (rest, paracetamol); oral acyclovir for eczema herpeticum and varicella zoster.

Bacterial

Most are either *Staphylococcus* or *Streptococcus* – treat with penicillin (erythromycin if penicillin allergic).

Mycobacterial

Leprosy; buruli ulcer – surgical (no effective medical treatment).

Fungal

Oral griseofulvin ◈ (tinea capitis, widespread corporis); selenium sulphide shampoo (tinea capitis, versicolor); ketoconazole cream (tinea versicolor); imidazole cream (tinea corporis); continue treatment up to a week after symptoms resolve ◇.

Nappy rash

Allow skin to breathe; 0.5%–1% hydrocortisone for three days, plus emollient; consider nystatin for fungal supra-infection.

Critical documentation

Use a body map to document distribution of widespread rash. Record time course of rash, and recommended treatment. Be sure to document presence or absence of systemic signs or symptoms, including fever.

Disposition

Admit acutely unwell patients as needed. Most cases can be followed up in a primary care setting. Consider dermatology or paediatric referral if unsure.

60 Approach to vesicular-bullous rash

Many conditions present with a vesicular or bullous rash. These lesions may represent a relatively innocuous disease (herpes simplex) or a life threatening condition (epidermolysis bullosa, toxic epidermal necrolysis (TEN) etc.).

The first five minutes

ABC, VS, O2; IVF as needed. Contact precautions.

History and physical examination

Key historical features

Onset, distribution and evolution over time; age and gender, family history of similar eruption (epidermolysis bullosa); sexual history (herpes simplex). Check vaccinations, past medical history and medications (drug eruptions); infected contacts (varicella, coxsackie). Check for associated symptoms, especially fever.

Signs and symptoms

Assess overall condition to include VS, blood glucose and mental status. Inspect skin, hair, nails, oral/genital/conjunctival mucosa. Note associated erythema, urticaria or erosions. Characterise blisters by their size, location and distribution. Systems review to include oral, ocular, genitourinary, GI and respiratory.

Possible causes and differential diagnosis

- *Infant*: epidermolysis bullosa, SSSS
- *Child:* varicella (chicken pox), coxsackie (hand-foot-and-mouth)
- *Adult:* pemphigoid, pemphigus, zoster (shingles)

Associated symptoms

Pruritis (dermatitis herpetiformis, allergic contact dermatitis, papular urticaria (insect bites)), fever (varicella, coxsackie, erysipelas, SSSS), pain (zoster), presence of specific triggers/allergens, heat (burns).

Investigations

Most presentations are diagnosed clinically. Use investigations as directed by systemic illness.

Management

The goal of acute management is to treat pain and itching, provide wound care, evaluate and treat systemic illness, and find and reverse the cause.

General

- Varicella (📖 ch. 132)
- Coxsackie: conservative, control fever

• Uncomplicated: treat with soap, water, clean dressings
• Infective: topical treatment (systemic/oral for poor response)
• Analgesia, skin protection
• Large skin surface area involvement: treat like burns; hydrate

Critical documentation

Distribution of rash and recommended treatment; presence of systemic signs, including fever.

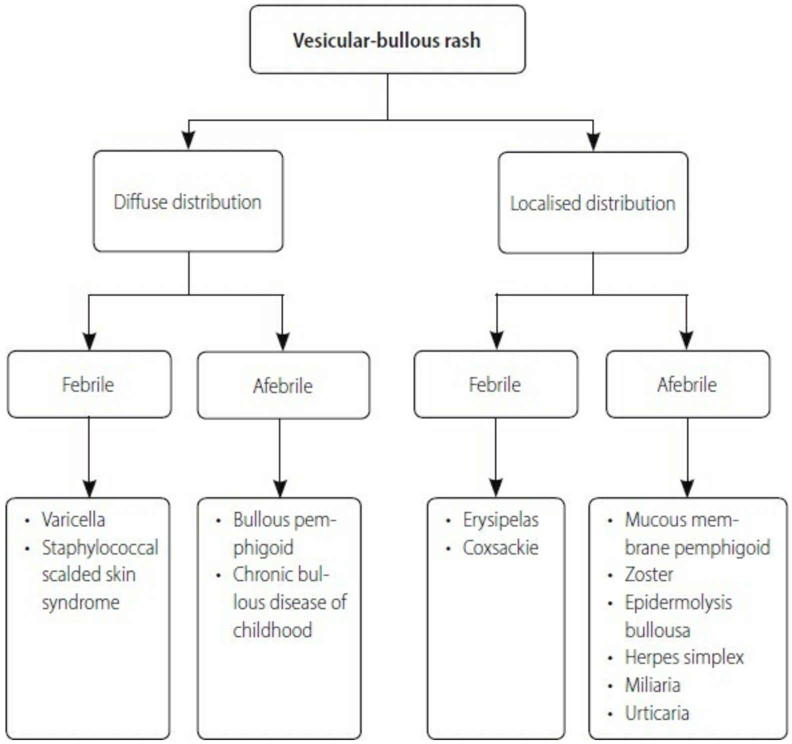
Disposition

Admit acutely unwell patients. Most cases can be followed up in a primary care setting. Consider dermatology referral if unsure, and for all diffuse bullous conditions.

Table 60.1 Causes, presentation and management

Primary rash		
Condition	**Presentation**	**Management**
Bullous pemphigoid	Auto-immune bullous disease; trunk, proximal limbs, skin folds; clear, cloudy or bloody	Prednisolone
Mucous membrane pemphigoid	Auto-immune bullous disease; oral lining; elderly women	Topical steroids. Short burst prednisolone. Eye, oesophagus, trachea, larynx involvement may require immunosuppressants ◇
Epidermolysis bullousa	Inherited connective tissue bullous disease; infants; friction, minor trauma sites, hands , feet	Avoid blister formation (soft clothes, sheep skin bedding, etc.)
Chronic bullous disease of childhood (CBDC)	Vesicular-bullous; children; starts genital area, spreads to hands, feet, face	Dapsone or sulphapyridine
Pemphigus vulgaris	Auto-immune bullous disease; mostly mucosal; rare	Fluids/electrolytes. high dose prednisolone
Secondary rash		
Condition	**Presentation**	**Management**
Varicella	Vesicular; children; lesions in different stages of development; follows flu-like illness	
Erysipelas	Bullous; superficial cellulitis; lower limbs; butterfly cheeks	Penicillin 10–14 days
Coxsackie	Vesicular; children; hands, feet and mouth; associated with flulike illness with high fever	
Herpes simplex	Vesicular; lips, genitals. Pain/burning may precede visible lesions.	Topical acyclovir, famcyclovir or valacyclovir. Systemic treatment for disseminated, CNS, eye, immunocompromised or systemic involvement ◇
Zoster	Vesicular; adults, immune-compromised; pain may precede visible lesions	Antivirals as above
Bullous impetigo	🕮 Impetigo, p. 160	
SSSS	Bullous; children; scald-like blisters, systemically unwell	Fluids/electrolytes, flucloxacillin
Frostbite/burn	Vesicular-bullous; associated thermal exposure	
Miliaria	Vesicular; heat/sweat rash	
Papular urticaria	Vesicular-bullous; localised crops; seasonal	
Porphyria cutanea tarda	Vesicular-bullous; fragile skin, hand dorsum and forearms ; dark urine	
Drug eruptions	🕮 p. 165	

Figure 60.1 Algorithmic approach to the emergency diagnosis of the vesicular-bullous rash



Adapted from Murphy-Lavoie H. Emergent diagnosis of the unknown rash, the algorithmic approach. *Emergency medicine* 2010:42(3):6–17

61 Approach to purpura

Petechiae, purpura and ecchymosis are skin lesions caused by haemorrhage of the small blood vessels of the skin (leukocytoclastic). Lesions are dark, red-purple, do not blanch on pressure and may be palpable. Petechiae are 1–3 mm, purpura are larger, and ecchymoses are large areas of confluent purpura. A purpuric rash should always be taken seriously and may represent a rapidly-progressive life-threatening illness. Unlike other rashes, even very few isolated purpura may represent a life threatening condition.

The first five minutes

ABC, VS, O_2; IVF as needed.

Consider full contact precautions and isolation if viral haemorrhagic fever (VHF) suspected. Concern for meningococcal disease warrants droplet precautions (standard PPE and mask).

History and physical examination

Key historical features

Timing of onset and progression of lesions; associated or recent systemic illness especially GI or CNS symptoms, drug history or access to anticoagulants, abnormal bleeding history (easy bruising, gum bleeding, epistaxis, or menorrhagia) and any family history of bleeding disorders. Also ask about travel, especially to areas where VHF are endemic, and recent tick bites.

Signs and symptoms

Evaluate for systemic illness. Note lesion size, confluence, whether palpable and if any associated vesicular lesions and their contents (exudate, blood, pus) (📖 Describing skin lesions, p. 142). Note distribution of purpura on body (limited to upper body, or lower extremities). Check for mucous membrane involvement.

Possible causes and differential diagnosis

Usually associated with infection (meningococcaemia, endocarditis, VHF, tick bite fever), immune diseases and vasculitis (ITP, TTP, HSP), coagulopathies (haemophilia, vWd, DIC, anticoagulant overdose) or trauma.

Meningococcaemia: petechial rash progresses over minutes to hours (📖 Meningitis, p. 330).

TTP: more common in women 30–50 years. Patients usually ill. Pentad of microangiopathic haemolytic anaemia, thrombocytopaenic purpura, neurological abnormalities, fever, and renal disease. Often look ill.

ITP: acute form peaks in ages 2–4 years and chronic form in ages 20–50 years; female predisposition for ages > 10 years. Auto-immune destruction of platelets. Patients usually well-appearing.

Disseminated gonococcaemia: maculopapular rash to purple, haemorrhagic vesicles, sparse, tender, peripheral (📖 Sexually transmitted infections, p. 356).

Endocarditis: petechial rash, fever, tachycardia, murmurs and/or failure (📖 Endocarditis p. 100).

HSP: mainly children, purpuric rash typically on buttocks and lower limbs, often associated with abdominal pain, arthralgias, oedema.

Vasculitis: palpable purpura (may ulcerate), usually affecting lower limbs predominantly.

Tick-bite fever: tick bite eschar, fever and malaise, headache, maculopapular to purpuric rash often involving the palms and soles (📖 p. 376).

Lesions limited to the face, neck and upper trunk (SVC territory), may result from forceful coughing or vomiting.

Investigations

Labs: urinalysis (haematuria/proteinuria in (HSP, endocarditis, vasculitis), CBC (low platelets in ITP, TTP, DIC; low Hb in TTP/ endocarditis; high WBC in infections), creatinine (elevated in dehydration, TTP, HSP), blood culture ◇; culture of scrapings of the lesions if sepsis, PT/PTT (DIC, meningococcaemia) ◈.

Specific testing

- TTP: schistocytosis on peripheral smear, raised LDH/ bilirubin ◇
- HSP: lipase (or if not available, amylase) to rule out pancreatitis ◈

Management

The goal of acute management is supportive care, early diagnosis of life-threatening conditions, and limiting transmission (isolation as needed). Do not delay empiric antibiotic treatment for diagnostic testing if systemic bacterial illness suspected.

General

Toxic patients: ABC, O2, IVF, ECG monitor, blood glucose, stat IV/ IM dose of penicillin G OR third generation cephalosporin (ceftriaxone or cefotaxime).

Specific

📖 Meningitis, p. 330; STI, p. 356; Endocarditis, p. 100; Tick bite fever, p. 376, Coagulopathies, p. 288.

- TTP: Treat underlying cause, consider plasma exchange, steroids. Avoid platelet transfusion unless uncontrolled haemorrhage ◈. Involve a specialist early.
- ITP: for severe haemorrhage consider platelet transfusion if risk of major bleeding, steroids (hydrocortisone IV), IV immunoglobulin ◈. Usually spontaneous resolution in weeks to months
- Vasculitis: 📖 p. 648.

Critical documentation

Document distribution and progression of lesions. Presence of systemic signs, including fever. Note timing of presentation, serial exams of lesions and timing of treatments administered. Note any travel history or tick exposure.

Disposition

Admit all patients with purpuric rash and systemic illness. TTP/ITP: Early haematology consultation ◈.

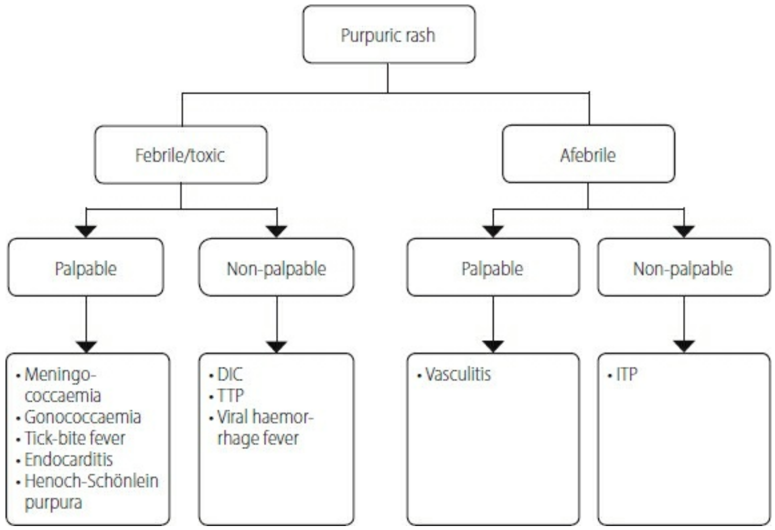


Figure 61.1 Diagnosis of the unknown rash

Adapted from Murphy-Lavoie H. Emergent diagnosis of the unknown rash, the algorithmic approach. *Emergency medicine* 2010:42(3):6–17

62 Pediculosis and scabies

Pediculosis and scabies are caused by infestation with parasitic lice or mites, are characterised by intense itching, and can be very contagious.

Pediculosis

Lice infestation. Eggs (nits) are cemented to hair or clothing and hatch in 6–10 days. The 1 mm egg case then turns white.

- Pediculosis capitis (head lice): scalp hair, 1–2 mm length
- Pediculosis pubis (pubic lice, ‘crabs’): pubic hair, 1 mm length
- Pediculosis corporis (body lice): fabrics/clothing (but live off host), 2–4 mm length

Scabies

Sarcoptes scabiei mite burrows under the outside layers of the skin, typically of the hand.

The first five minutes

Careful attention to infection control. Isolate the patient; notify staff of the need for contact precautions.

History and physical examination

Key historical features

Pediculosis

Mainly an itchy scalp, pubic area or body.

Scabies

Severe persistent itch, worse at night. Rash in the affected areas.

Signs and symptoms

Pediculosis

Closely examine the affected area; a magnifying glass is useful. The lice are difficult to see; look for dark specks of louse faeces, nits, adult lice, and excoriations from scratching. Lymphadenopathy with secondary infection. Blue-grey macules (maculae cerulean) with pubic and body lice bites.

Scabies

- *Burrows*: thin, serpiginous, 2–3 mm long lines, often with scaling, excoriation, redness, hyperpigmentation, ends with vesicles. Typically on finger web spaces, flexor aspects of wrists; can affect other areas (face, scalp and perineum – infants /bedbound elderly)
- *Generalised rash*: tiny red, itchy bumps on limbs and trunk
- *Nodules*: itchy nodules in armpits, groin or penis shaft
- *Blisters/pustules*: affects palms and soles in infants

Crusted (Norwegian) scabies

Massive, generalised infestation with millions of mites. More common in immunocompromised patients, and has occurred as a nosocomial outbreak in ICU settings. Crusting with thick, hyperkeratotic scales over knees, elbows, soles and palms. Varying degrees of itching.

Possible causes and differential diagnosis

The differential includes any pruritic rash.

Pediculosis

Head lice – seborrhoeic dermatitis, dandruff, dermatophytes; *pubic lice* – folliculitis, contact dermatitis, dermatophytes; *body lice* – dry skin, impetigo, scabies, insect bites.

Scabies

Scabies: insect bites, atopic dermatitis, contact dermatitis, urticaria, syphilis. *Norwegian scabies* – psoriasis.

Investigations

Both are clinical diagnoses.

Pediculosis

If unsure, consider microscopy; Woods lamp fluoresces lice ◇. Other STI screening if pubic lice.

Scabies

Microscopic examination of mites, eggs, fragments (skin scraping sample) on mineral oil preparation if unsure ◇.

Management

The goal of acute management is to provide symptomatic relief, eradicate the infestation and limit transmission and supra-infection.

In addition to treatment below, wash bedding, towels and clothing (60 °C); wear clean clothing after skin treatment. Re-treat in 7–10 days. It is essential to treat all household members and close personal contacts. Itching may persist 1–2 weeks after successful treatment – treat with antihistamines. Treat secondary infections with appropriate antibiotics. For pediculosis, remove eggs from wet hair with nit comb; use petroleum ointment for eyebrow infestation.

- Permethrin 5% cream: first line, apply from the chin down, wash off after 8–14 hours, single application (not for infants < 2 months)

- Lindane: second line, apply over entire body excluding face, wash off after 8–12 hours (potentially neuro-toxic, only use if failed permethrin) ◇
- Ivermectin: single 200 mcg/kg oral dose (is as effective as permethrin); most effective with encrusted scabies, immunocompromised patients, or when topicals are contraindicated ◇
- Ivermectin lotion: single treatment to hair; leave 10 minutes, then wash (for adults and children > 6 months) ◇ (pediculosis only)
- Malathion 5% cream: apply to hair 8–12 hours, wash (pediculosis only)
- Benzyl alcohol 5% lotion: apply to hair, 10 minutes then wash (pediculosis only)
- Benzyl benzoate 25% emulsion: apply over entire body three times in 12 hour intervals, avoiding eyes/mucous membranes. Wash off 12 hours after last application. (Avoid in pregnancy, neonates, or broken skin.) ◇ (scabies only).
- Sulphur 6%: for neonates or pregnant/breastfeeding women; inexpensive; apply chin down, wash off with soap/water after 24 hours, successive three day treatments ◇ (scabies only)

Critical documentation

Distribution of rash, and recommended treatment; presence of systemic signs or symptoms.

Disposition

Admit acutely unwell patients and patients with Norwegian scabies. Most cases can be followed up in a primary care setting. Consider dermatology referral if unsure.

63 Impetigo

An acute, superficial skin infection caused by either *Staphylococcus aureus* and/or group A beta-haemolytic *Streptococcus* (GABHS, also called *Streptococcus pyogenes*), which is highly contagious. Impetigo is the most common skin infection in children.

The first five minutes

ABC, VS, IVF as needed.

History and physical examination

Key historical features

Often follows minor skin trauma, insect bites, scabies, herpes simplex, varicella, or eczema; contact with an infected person often reported. Ask about haematuria and urine output (acute post-streptococcal glomerulonephritis (APSGN) is a rare complication). Ask about comorbid conditions, allergies and medication. Make sure the child is feeding normally.

Signs and symptoms

General

Determine if the child is well or ill – systemic involvement may lead to sepsis and peri-oral lesions may interfere with feeding, leading to dehydration and, in the very young, hypoglycaemia. Check for glomerulonephritis.

Non-bullous impetigo (impetigo contagiosa)

Develops from a single macule to a vesicle/pustule with golden crusting on a red base. Many lesions may be present. Surrounding erythema is rare. Most often affects the face (mouth or nose) or periphery (palms and soles spared). Systemic involvement is unusual (regional lymphadenopathy common).

Bullous impetigo

Thin-roofed bullae (< 3 cm) containing clear, straw-coloured fluid; on rupture leaves a moist erythematous base with a scaly rim. Common in neonates and affects the face, trunk, extremities, buttocks, or perineum. Patients are usually systemically well, but may have fever, diarrhoea or weakness. Check for signs of dehydration.

Possible causes and differential diagnosis

Non-bullous impetigo: herpes simplex, varicella zoster, tinea corporis (which does not affect the upper lip), cutaneous candidiasis (often associated with burning/pruritus), pediculosis (isolated to the scalp), scabies and eczema/dermatitis. Consider MRSA in endemic areas.

Bullous impetigo: pemphigus vulgaris, early SJS, bullous lupus (rare) or bullous scabies (rare).

Investigations

History and examination are often diagnostic. Use investigations when the diagnosis is unclear or there is concerns for sepsis, dehydration, hypoglycaemia or APSGN.

- Labs: urinalysis ◇ (haematuria and/or proteinuria may suggest APSGN) (📖 Post infectious conditions p. 382)
- Swab ◇: swab lesions after washing away the crust and lifting the scab

Management

The goal of acute management is to identify and treat dangerous complications, provide symptomatic relief, treat infection and limit transmission.

- Prevent spread to others: encourage good hand hygiene, avoid sharing towels or ointments, and wash and change clothes, towels and bed sheets frequently
- For mild infections dress or bathe affected areas with saline, betadine, or potassium permanganate (avoid on the face – causes staining)
- Avoid petroleum jelly products
- Cloxacillin 7–10 days for severe infections (erythromycin if penicillin allergic)
- Consider topical mupirocin for *S. aureus* infections
- Manage APSGN

Critical documentation

Document distribution of rash, and recommended treatment. Be sure to document presence or absence of systemic signs or symptoms, including fever.

Disposition

Admit acutely unwell or dehydrated patients. Most cases can be followed up in a primary care setting. Dermatology or paediatric referral if unsure.

64 Dermatoparasites

Parasite infestation of the skin is more common in warm, humid environments, and may be typical of a specific area as a result of parasite habitat. The vast majority of parasitic infestations have a slow onset with the time between vector transmission and symptoms delayed by weeks to months (sometimes years as with onchocerciasis).

The first five minutes

- ABC, VS, O_2, IVF as needed
- Remove clothes for inspection, but avoid hypothermia. Always wear personal protective equipment

History, physical examination and differential diagnosis

Most parasites have a long delay from exposure to presentation. Exceptions are creeping eruption (larva migrans) which may result in an eruption at the transmission site within 30 minutes, and cutaneous leishmaniasis which can

result in a typical sore within days of sandfly vector transmission.

Conditions usually present with a combination of pain, itching and visible skin lesions.

Condition	Parasite	Presentation	Other characteristics	Diagnostics
Creeping eruption (larva migrans)	Hookworm transmitted from walking, working or sitting in infested soil	2–3 mm wide snake-like, itchy tracks usually to feet (thighs and buttocks in small children)	Humans are accidental, dead-end hosts	Clinical diagnosis
Cutaneous leishmaniasis (sore type)	*Leishmania tropica*, major, infantum or aethiopica through female sandfly vector	Starts as one or more red papule (2 cm), usually face or extremities which darken and then ulcerate	Tends to leave a scar when healed; mainly North Africa	Clinical diagnosis or skin snip biopsy (70% yield) ◇
Mucocutaneous leishmaniasis	*Leishmania aethiopica* through female sandfly vector	As above, but occurs around mucosa of nose and mouth. May destroy tissue and disfigure	Mainly Ethiopia and Kenyan highlands	Clinical diagnosis or tissue biopsy ◇
Diffuse cutaneous leishmaniasis	*Leishmania aethiopica* through female sandfly vector	Starts as nodule on face or limb extensor surfaces which slowly disseminates. Nodules do not typically ulcerate	Often mistaken for lepromatous leprosy; Ethiopia and Kenyan highlands. Cure is difficult	As above
Visceral leishmaniasis (kala azar)	*Leishmania donovani* or *infantum* through female sandfly vector	Scaly, gray, dark, ashen skin associated with fever, weakness, diarrhoea, emaciation, swollen glands and hepatosplenomegaly	Can be life threatening; mainly Kenya and North Africa	As above, or serology ◈, plus blood count and liver function tests ◇
Lymphatic filariasis	Roundworm (*Wuchereria bancrofti*) transmitted through mosquito vector	Thickening of skin and swelling of subcutaneous tissue of limbs, breasts, genitalia (elephantiasis)	Result from immune response to dying worms in lymphatics	Clinical diagnosis, blood smear ◇ or antigen/antibody testing of blood ◈
Onchocerciasis (river blindness)	Onchocerca volvulus transmitted through blackfly vector	Itchy, red, spotty rash on mainly lower trunk, pelvis, buttocks, thighs and legs, leading to thickened, dry skin. Firm, flattened/bean-shaped, movable, non-tender nodules on skull, ribs, scapulae, elbows or knees	Can become chronic leading to permanent scarring and lizard-like skin. Eye involvement may cause blindness	Clinical diagnosis or skin biopsy ◇. Wriggling microfilariae may be visible by slit lamp in anterior chamber of eye ◈

Management

Creeping eruption (larva migrans)

- Usually self-limiting, but experienced clinician may consider freezing the skin with chlorethyl spray, liquid nitrogen, or solid carbon dioxide 1 cm ahead of visible trail
- Albendazole 400 mg PO daily for 3 days OR ivermectin 200 mcg/kg orally once OR topical thiabendazole 10–15% until 2 days after burrows disappear. Can also crush three 400 mg tablets of albendazole in 12 g of petroleum jelly, apply TID for 10 days

Leishmaniasis

- Itraconazole or ketoconazole sometimes effective
- For sore type consider cryosurgery or excision for lesions that fail to spontaneously resolve ◇
- For diffuse cutaneous, mucocutaneous or visceral type supportive care and either of the following:
  - » Stibogluconate (Glucantine or Pentostam) 20 mg/kg/day IV or IM for 3–4 weeks.
  - » Amphotericin B 1 mg/kg IV alternate days for two months, up to 3 g total

Lymphatic filariasis

Stress local hygiene; promote lymph flow though exercises, lymph-massage, intermittent compression stockings or bandages; ivermectin and albendazole single dose repeated annually for 3–4 years.

Onchocerciasis

Ivermectin single dose repeated every 3–12 months while symptomatic with itching.

Critical documentation

Use a body map to document distribution of widespread lesions. Record VS and treatment given. Consider tracing contacts if highly contagious.

Disposition

Most patients can be discharged for out-patient follow-up. Provide advice on prevention (avoiding dusk and dawn when sandflies are most active, covering up with clothing, use of insect repellents and sleeping nets).

Admit severe visceral leishmaniasis.

65 Drug eruptions

Rashes associated with drug therapy are common and may present in many forms. They range from benign (fixed drug eruptions), through irritating (drug-induced urticaria) to severe (Stephens-Johnson syndrome). Severe eruptions are often difficult to differentiate from infectious and other causes, and can be life threatening.

The first five minutes

ABC, VS, O_2; IVF as needed.

History and physical examination

Key historical features

Symptoms can start immediately or take weeks to develop after starting medications. Consider prescription, non-prescription, topical (including eye drops), vitamins and traditional medications. Note recently discontinued medications, date discontinued, and whether symptoms changed following discontinuation. Prior skin reactions to drugs or foods. Note whether there are systemic complaints, metabolic disorders, or immunocompromise.

Signs and symptoms

Most common drugs listed below,
* associated with serious reactions;
† drug reaction more common in patients with HIV.

Maculopapular exanthem (most common)

Symmetrical, confluent, reddish macules and papules (without palm or sole involvement), develops within two weeks after starting medication.

- ACE inhibitors, allopurinol*, amoxicillin, ampicillin, anticonvulsants*†, barbiturates, cetirizine, *Ginkgo biloba*, hydroxyzine, isoniazid, nelfinavir, NSAIDs*, phenothiazine, quinolones, sulphonamides*†, thalidomide, thiazides, trimethoprim-sulfamethoxazole*†

Hypersensitivity syndromes

Eczema, uticaria, scaling, weepy blisters erythema, papules (sometimes vesicles and pustules). These may occur in the form of classic allergic conditions or more severe systemic presentations, including anaphylaxis or systemic hypersensitivity associated with high fever (usually before rash), inflammation of one or more systems (including hepatic, renal, respiratory and/or cardiac) and eosinophilia. Typically develops around 1–3 weeks after starting medication although may take up to three months.

- Allopurinol*, amitriptyline, anticonvulsants*†, dapsone, lamotrigine, NSAIDs*, olanzapine, phenobarbital, saquinavir, spironolactone, sulphonamides*†, zalcitabine and zidovudine

Stevens-Johnson Syndrome (SJS)

Extensive skin involvement with initial large but atypical targetoid lesions, followed by erythema, blistering and skin loss. Involvement of mucous membranes and ≥10% of the skin distinguishes SJS from erythema multiforme. Develops within a few days to a month after starting medication. Mortality 5%. This condition is significantly more common in patients who are HIV positive and taking antiretrovirals, TB treatment or bactrim.

- Allopurinol*, aspirin/NSAIDS*, anticonvulsants*†, barbiturates, cimetidine, ciprofloxacin, codeine, didanosine, diltiazem, erythromycin, furosemide, griseofulvin, hydantoin, indinavir, imidazole antifungals, nevirapine, nitrogen mustard, penicillin†, phenothiazine, phenylbutazone, ramipril, rifampicin, saquinavir, sulphonamides*†, tetracyclines and trimethoprim-sulfamethoxazole*†

Toxic epidermal necrolysis (TEN, severe form of SJS)

Sunburned sensation, followed by rapid, generalised, full skin thickness sloughing, affecting ≥30% of skin (mortality is around 30%, and HIV patients are at higher risk).

- Alfuzosin, allopurinol*, aspirin/NSAIDs*, anticonvulsants*†, sulfadoxine and pyrimethamine (Fansidar), isoniazid, lamotrigine, lansoprazole, letrozole, penicillin†, prazosin, sulphonamides*†, tetracyclines, thalidomide, trimethoprim-sulfamethoxazole*† and vancomycin

Differential diagnosis

Drug eruptions may mimic many skin conditions and the differential is wide.

- Erythema multiforme: self-limiting rash caused by infection in 90% of cases (most often herpes simplex). Typical target lesions (dark/crusted centre and outer ring around a paler raised middle ring) start peripherally and spread to trunk; mucosal involvement usually involves the lips.
- Erythroderma (generalised exfoliative dermatitis): scaling and generalised erythematous dermatitis involving 90% or more of the skin
- Contact dermatitis: pruritic papules and vesicles on an erythematous base caused by reaction to chemicals. Distribution localised to contact area

Investigations

History and examination are diagnostic in most cases; reserve investigations for severe eruptions.

- Labs ◇: CBC (abnormal WBC/platelets and eosinophilia in severe eruptions), creatinine, electrolytes, ABG, LFTs
- Swab ◇: culture may be required to diagnose site infection

Management

The goal of acute management is symptomatic treatment, addressing any systemic illness and discontinuing the offending medication.

Exanthem

Oral antihistamines (chlorpheniramine, promethazine, etc.) are first line, use Calamine lotion with or without menthol 0.25% and/or phenol 1%, or zinc oxide cream for pruritus or weepy lesions. For more severe reactions, start oral prednisolone at 40–60 mg and taper over two weeks ◇.

SJS and TEN

Inpatient supportive treatment. Strict infection control; wound dressing, temperature control and fluid and electrolyte management. TEN is best treated in a specialised dermatology or burns unit. Fluid requirements as for burns (📖 Burns, p. 776); monitor electrolytes and urine output. Intravenous immunoglobulin may improve outcome (controversial) ◈.

Hypersensitivity syndrome

Supportive care, consider systemic corticosteroids. Involve a specialist early.

Critical documentation

Full drug history, distribution of rash, recommended treatment. Be sure to document presence or absence of systemic signs or symptoms, including fever.

Disposition

Admit acutely unwell patients. Admission for fluid resuscitation is required for dehydrated patients, especially children. Most cases can be followed up in a primary care setting. Consider dermatology referral.

Part

4

D. Ear, nose and throat

66 Life-threatening ENT infections

The head and neck represents an extremely high density of organs and structures – many essential to life (i.e. airway, great vessels, upper spinal cord etc.). Any infection of the head and neck may threaten life, most often by compression and occlusion of the airway. Early recognition and intervention is essential.

Pay particular attention to:

- Airway compromise: voice change, noisy breathing, respiratory distress
- Vascular involvement: stroke-like presentation
- Mediastinal involvement: sepsis, chest pain, dysphagia
- Intracranial spread: focal deficits, meningism, AMS
- Systemic sepsis

The keys to successful management are: early recognition, early aggressive airway control, rapid initiation of therapy and early involvement of the definitive care team.

Clinical features and management

Table 66.1 Clinical features and management of life-threatening ENT infections

Condition	Clinical features	Investigations	Management
Croup (📖 p. 174)	Usually child 1–6 years Tachycardia Fever Tachypnoea Barking cough Stridor	Clinical diagnosis	O_2 by mask Adrenaline nebulisation Steroids Intubation if required
Epiglottitis (📖 p. 176)	Sitting patient Fever Sore throat Dysphagia Drooling Stridor Sepsis	Lateral neck XR CT scan	O_2 by mask Definitive airway with gaseous induction IV antibiotics – 3rd generation cephalosporin
Peri-tonsillar abscess	Sitting patient Fever Sore throat Dysphagia Drooling Stridor Sepsis Visible swelling and inflammation of the tonsils and surrounds	Lateral neck XR	O_2 by mask Needle aspiration Consider steroids Potential definitive airway though intubation rarely required IV antibiotics – 3rd generation cephalosporin Aspiration or incision and drainage
Retropharyngeal abscess	Fever Dysphagia Sore throat Voice change Abnormal neck posture	CT scan Endoscopy Lateral neck XR	Potential definitive airway Rapid referral IV antibiotics – 3rd generation cephalosporin Aspiration or incision and drainage
Mastoiditis	History of otitis media Pain and fever Hearing loss Discharge from middle ear Headache Redness behind ear	Clinical diagnosis CT scan	Pain control IV antibiotics (e.g. cefotaxime) Analgesia Incision and drainage Neurosurgical intervention for intracranial spread
Mediastinitis	History of chest surgery or endoscopy Chronic illnesses (TB, sarcoid, malignancy) Radiation Chest pain Fever/chills Dyspnoea	CXR CT chest Mediastinoscopy	O_2 by mask IV antibiotics (start with cefotaxime or equivalent; adjust to culture results) Surgery ICU admission
Cavernous sinus thrombosis	Fever Headache Photophobia Bulging eye unilaterally Cranial nerve abnormalities	CT brain	O_2 by mask IV antibiotics (e.g. cefotaxime) Early neurosurgical admission and intervention
Sinusitis	High fever Purulent nasal discharge Altered sensorium Meningism	Sinus XR CT Aspiration and culture of sinuses	Antibiotics, analgesia, decongestant Surgical drainage if required

Disposition

Admit all patients with potential airway compromise to the highest available level of care. Admit systemically ill

patients.

67 Approach to stridor

Stridor is an abnormal, high-pitched sound produced by turbulent airflow through a partially obstructed airway at the level of the large extrathoracic airway (supraglottis, glottis, subglottis, and/or trachea). It is a symptom not a diagnosis and the underlying cause must be determined. Stridor is **always a dangerous finding** and may indicate imminent airway obstruction.

Stridor may be inspiratory, expiratory, or biphasic. Inspiratory stridor suggests a laryngeal obstruction, while expiratory implies tracheobronchial obstruction; biphasic suggests a subglottic or glottic anomaly.

The first five minutes

Examine the patient in their position of comfort. Avoid agitating children with stridor. Avoid unnecessary and rough examination of the upper airway. Assess severity of airway obstruction, and work of breathing. Loudness of stridor does not correlate with severity of obstruction; persistent increased work of breathing, and stridor when calm suggests significant obstruction.

- ABC, VS; if hypoxic, facemask O_2 as tolerated; IV access as tolerated
- If moderate or severe distress at rest give nebulised adrenaline 0.5 ml/kg of 1:1 000 solution (max 5 ml) and steroids
- Prepare for emergency intubation as needed (in a high care area, the most experienced doctor, difficult airway and surgical airway equipment ready)

History and physical examination

Key historical features

Ask about onset, severity, fluctuation and duration. Also ask about similar episodes in the past. Ask about features that may suggest the cause (i.e. a viral prodrome, high fever and odynophagia, smoking, alcohol use) and existing medical conditions (head and neck malignancies, airway abnormalities, previous airway injuries or surgery). Any foreign body aspiration?

Infectious causes are suggested by history of cough, fever or sore throat.

Signs and symptoms

The critical initial step is to determine if there is danger of losing the airway. Important red flag findings include: hypoxia, respiratory distress, AMS, high work of breathing, exhaustion, inability to speak, inability to swallow.

Other features to evaluate: fever, features of upper airway infection, abnormal pulmonary findings (wheeze, crackles, collapse) etc. Perform a complete head, neck and ENT examination.

Investigations

Initial investigation of stridor is clinical. Special investigations will depend on the suspected diagnosis.

- Imaging: lateral neck XR (FB, soft tissue swelling, masses), CXR (FB, evidence of aspiration) ◇; CT (extent and cause), endoscopy (carefully attempted by a specialist in theatre with full resuscitation and surgical airway equipment) ⟐

Differential diagnosis and possible causes

- Infectious causes include croup, epiglottitis, peri-tonsillar abscess, retropharyngeal abscess
- Non-infectious causes include FB obstruction, burns, trauma (internal or external), allergic reactions/anaphylaxis, bites and stings, and malignancy

Treatment

The goal of acute management is to identify and treat a threatened airway and to ensure oxygenation. Consider early

adrenaline while the cause is being sought. Further management depends on the cause.

- General: keep the patient calm, humidified O_2, allow the patient to assume their most comfortable position (usually sitting)
- Specific:
  - Infectious: consider:
    - Nebulised adrenaline 0.5 ml/kg of 1:1 000 and steroids (single dose dexamethasone 0.6 mg/kg PO OR prednisolone 1–2 mg/kg PO)
    - IV antibiotics
    - Drainage of abscesses
  - Non infectious:
    - Treatment should be directed at airway support and maintenance
    - Medication for anaphylaxis 📖 p. 38)

Critical documentation

Note clinical findings, investigation results, treatment and response.

Disposition

Admit ill patients with stridor as the main complaint, those in whom diagnosis is not clear, and those who fail to respond after 4–6 hours of observation. Discharge other patients after 4–6 hours of observation.

68 Croup

Croup is an acute viral respiratory illness causing inflammation of the subglottic region of the larynx. It is most severe in children < 3 years.

The first five minutes

Adopt a hands-off approach. If possible, allow child to remain on carer's lap. Do not examine the throat – can lead to complete airway obstruction. If hypoxic, attempt facemask O_2 if tolerated

Assess severity of airway obstruction, and work of breathing. Loudness of stridor does not correlate with severity of obstruction; persistent increased work of breathing, and stridor when calm suggests significant obstruction.

If moderate or severe distress at rest: nebulised adrenaline 0.5 ml/kg 1:1 000 (max 5 ml) and steroids.

History and physical examination

Key historical features

Noisy breathing, barking cough, hoarse voice, difficulty in breathing, fever. Onset either sudden or after a period of coryza.

Signs and symptoms

Severe croup

Any combination of:

- **Severe respiratory distress** – chest indrawing, alae nasae flaring, grunting, tachypnoea, low O_2 saturation
- **Stridor** – maybe inspiratory or bi-phasic (i.e. inspiratory + expiratory)
- **Cyanosis** – bluish-grey lips/tongue and/or pallor
- **Pulsus paradoxus** – pulse weakens or disappears during inspiration
- **Exhaustion** – agitation of ↓ level of consciousness

Beware – the exhausted child may have paradoxical signs due to ↓ respiratory effort.

Moderate croup

- NO signs of severe croup
- Mild-moderate respiratory distress and/or stridor present at rest
- Signs worsen when active

Mild croup

- NO signs of severe croup
- Mild stridor ± mild respiratory distress NOT present at rest
- Signs only present when upset or active

Possible causes and differential diagnosis

- Bacterial upper airway infections (e.g. epiglottitis, bacterial tracheitis) – sudden onset, toxic appearance, high fever, drooling, tripod posture. (See also 🕮 p. 176, 🕮 p. 170)
- Foreign body – sudden onset of coughing or choking, young infant, often during play or eating (see 🕮 p. 184)
- Acute anaphylaxis – allergen ingestion or insect bite/sting (🕮 p. 38)

Investigations

Croup is a clinical diagnosis; no confirmatory investigations are required.

Management

The goal of acute management is to correct hypoxia and improve airflow.
Give facemask O_2 if tolerated; use minimal invasive procedures.

Severe

Adrenaline ◇:
- Nebulised adrenaline 0.5 ml/kg of 1:1 000 solution (max 5 ml)
- Repeat every 10–15 minutes or continuous *in extremis*
- Caution with repeated nebulisation: slow or stop nebulisation if extreme tachycardia develops
- If no nebuliser available – give IM/SC adrenaline 0.01 ml/kg of 1:1 000
- Impending respiratory failure – cautious intubation ◇, ideally with gaseous induction by anaesthetist

Steroids ◇:
- Single dose dexamethasone 0.6 mg/kg ◇ (oral, IM or IV)
- If no oral dexamethasone use IV dexamethasone solution orally
- If no dexamethasone prednisolone 1–2 mg/kg PO (repeat after 24 hours)

Moderate

- May need adrenaline nebulisation (see above)
- Oral dexamethasone 0.6 mg/kg single dose (or alternatives – as above)

Mild

- Oral dexamethasone 0.15 mg/kg single dose (or alternative – see above)
- Do not need adrenaline nebulisation or period of observation

All children with croup attending for emergency care should be given steroids.

Critical documentation

Note severity assessment, medication doses, timings, and response to treatment.

Disposition

Admit patients with severe croup, preferably to ICU ⟐. Discharge others, after six hours of observation if improved.

69 Epiglottitis

Epiglottitis is inflammation of the epiglottis and surrounding tissues, usually due to infection (the term is sometimes used to refer to chemical or physical inflammation). The primary concern is airway compromise: inflammation and swelling may rapidly occlude the airway, especially in children, leading to respiratory arrest and death.

The commonest bacterial causes are *H. influenzae* (children), *S. pneumoniae* (adults) and *S. aureus*. Viral causes include HSV and CMV. With increasing rates of *H. influenzae* vaccination in children, adult epiglottis cases are outnumbering those in children in many areas.

The first five minutes

ABC, VS, O_2, IV access, O_2 sat.

Allow the patient to assume a position of comfort. Defer procedures that may agitate the patient, as this may lead to airway obstruction.

History and physical examination

The cardinal symptom is a sore throat. Important complications include airway obstruction, local suppuration and systemic sepsis.

Key historical features

Ask about fever, URTI symptoms, viral prodrome (often absent). Rapid progression (within hours) is typical, especially in children. Adult cases may follow a more sub-acute course with prominent dysphagia (mimicking pharyngitis more closely).

Signs and symptoms

- Sore throat, dysphagia, foreign body sensation. Voice changes, drooling, stridor, respiratory distress
- Look for signs of airway compromise and systemic sepsis. Fever, tachycardia/tachypnoea, signs of shock, AMS; stridor, noisy breathing, respiratory distress
- Tenderness with gentle external palpation of the thyroid/hyoid or gentle rocking of the trachea (do NOT attempt in children, ill-appearing patients or those with any signs of airway compromise)
- Endoscopy or direct laryngoscopy should NEVER be attempted unless performed by an experienced clinician with full resuscitation, difficult airway and surgical airway equipment

Differential diagnosis

Bacterial or viral pharyngitis and tracheitis, retropharyngeal abscess, tonsillitis, peritonsillar abscess, croup (larygotracheobronchitis), anaphylaxis, angio-oedema, airway foreign body, laryngitis or any other cause or fever and airway compromise.

Investigations

Clinical examination is usually sufficient to make the diagnosis. Labs are of little use; consider in suspected sepsis. Consider imaging if clinical state allows.

- Imaging: neck XR (soft tissue swelling, often with a prominent epiglottis – the 'thumbs up' sign) ◇; CT (reveals inflammation and swelling, alternative diagnosis) ⟐

Treatment

The goal of acute management is to protect the airway and control systemic sepsis.

- Keep patient calm and allow to remain seated in a position of comfort
- Humidified O_2 by facemask if tolerated

- Airway management:
  - » Pro-active airway control is essential in patients with evidence of airway compromise and severe respiratory distress
  - » Anticipate difficult intubation: should be performed by an experienced doctor in theatre with full resuscitation, difficult airway and surgical airway equipment available
- Antibiotics (IV 3rd generation cephalosporin (ceftriaxone 1–2 g IV/IM 4–14 days) OR amoxicillin/clavulanic acid 1.2 g IV BID 10 days)
- Early referral to ENT

Nebulised adrenaline, corticosteroids, and beta-agonists have not been proven to be helpful.

Critical documentation

Record clinical and special investigation findings (particularly evidence of airway obstruction, respiratory distress and sepsis).

Disposition

Admit all patients epiglottis to the highest available level of care.

70 Pharyngitis and tonsillitis

Pharyngitis and tonsillitis are common inflammatory conditions of the throat. They are usually caused by infection (viral much more common than bacterial). Most cases are innocent and self-limiting. Important complications include local suppuration, airway compromise and post streptococcal glomerulonephritis.

The first five minutes

Usually not life threatening. Pay particular attention to airway, and manage as needed.

History and physical examination

Key historical features

The cardinal symptoms are a sore throat and dysphagia. Ask about immunocompromise, previous episodes or pre-existing medical conditions, medication and allergy.

Signs and symptoms

- Sore throat, dysphagia
- Fever, malaise
- Headache
- Viral pharyngitis is often associated with cough, runny nose, conjunctivitis, or diarrhoea
- Consider oral candida infection in immunocompromised patients
- Ask about ability to swallow, voice change, systemic illness and respiratory complaints

Signs include:

- Pharyngitis:
  - » Usually the only finding is an erythematous pharynx, possibly with reactive cervical lymphadenopathy
  - » An exudate or adherent membrane should raise suspicion of bacterial infection, candida or diphtheria
- Tonsillitis:
  - » Tonsillar swelling and redness (usually bilateral – unilateral involvement suggests bacterial infection, local complications or malignancy)
  - » An exudate is common
- Evaluate the ability to swallow and for evidence of dehydration
- Signs of systemic sepsis

• Signs of airway compromise

Differential diagnosis

• EBV infective mononucleosis
• Herpes pharyngitis
• Oral candida (consider in immunocompromised patients)
• Retropharyngeal abscess (unilateral swelling)
• Peritonsillar abscess (unilateral swelling)
• Epiglottitis (very ill-appearing, drooling)
• *Corynebacterium diphtheriae* (consider in unvaccinated patients)
• ENT malignancies

Investigations

Largely a clinical diagnosis, strep pharyngitis may be confirmed by a Strep rapid antigen screen ◈ or throat culture ◇.

Management

The goal of acute management is to exclude airway problems and identify streptococcal infection. Most cases of pharyngitis do not require antibiotics.

Symptomatic relief:
• Gargling with salt-water
• Analgesics and antipyretics

For severely symptomatic pharyngitis, consider single dose or short burst of steroid (dexamethasone 0.6 mg/kg to max 10 mg PO/IM/IV) ◇.

Treat all cases of confirmed or suspected streptococcal pharyngitis with antibiotics to prevent development of rheumatic fever.

• Penicillin is effective (penicillin V 500 mg PO TID 10 days. In patients <27 kg, 250 mg PO TID 10 days. Consider using single dose penicillin G IM 1.2 million units (<27 kg 600 000 units) ◇
• In patients with a penicillin allergy, use a first-generation cephalosporin or azithromycin, clarithromycin, or clindamycin ◇
• For oral candida, treat with nystatin (400 000–600 000 units PO four times a day) or fluconazole (200 mg PO daily) ◇

Critical documentation

Note airway compromise, pharyngeal exam (showing no evidence of retropharyngeal or peritonsillar abscess); ensure patient is able to drink fluids before discharge.

Disposition

Admit if concern for development of airway compromise, inability to drink adequate fluids, or if the patient is toxic-appearing. Discharge all other patients.

71 Epistaxis

Epistaxis is bleeding from the nostril, nasal cavity or nasopharynx. In true epistaxis the source of bleeding is the nose or nasopharynx. Bleeding from other sources, like the mouth or upper GI tract, may exit the nose, mimicking epistaxis.

It is the most common ENT emergency and may range from minor and innocent to severe bleeding with the potential for airway and haemodynamic compromise. Bleeding originates anteriorly at Kiesselbach’s plexus in 90% of cases. The most common causes are minor trauma and upper respiratory tract infections.

Consider the possibility of a secondary cause, such as coagulopathy or a nasal tumour, in all patients. Patients

presenting with recurrent or severe nose bleeds should prompt a more complete evaluation.

The first five minutes

Usually not life-threatening. Assess ABC and resuscitate as needed, paying particular attention to airway and haemodynamic compromise.

History and physical examination

Focus on three important questions:
1. Is there compromise of the airway?
2. Has the patient lost enough blood to cause haemodynamic compromise?
3. Has the patient lost enough blood to necessitate a blood transfusion?

Key historical features

• Ask about onset, duration, severity and previous episodes of bleeding
• Ask about features suggesting a secondary cause: easy bruising or mucosal bleeds, weight loss, nasal discharge or obstruction, trauma, URTI symptoms.

Remember to ask about existing medical conditions (particularly coagulopathy) and medications (aspirin/anticoagulants)

Signs and symptoms

Look for features of haemodynamic compromise.

When examining, use a face shield, gloves and a good light source. Provide the patient with a container to spit or vomit into and cover the patient with a towel or sheet.

Bleeding is usually obvious. Look for clots in the nose, blood trickling down the back of the throat and evidence of trauma. Attempt to visualise the source of the bleeding.

Look for evidence of a secondary cause (bruises, other mucosal bleeds, nasal tumours).

Investigations

Typically a clinical diagnosis; investigations may be needed in complicated cases or when a secondary cause is suspected (posterior epistaxis, ill patients, and those with suspected co-morbidities).
• Lab: CBC, type and cross ◇; PT/PTT ◈

Management

The goal of acute management is to arrest the bleeding and restore circulating blood volume (where needed).

Anterior epistaxis

• 1st line: positioning and pressure:
  » Position with seated with the head forward. Discourage swallowing of blood
  » Direct pressure for 15 minutes
• 2nd line: cautery:
  » Gently blow nose to remove clots then apply vasoconstrictive agents ◇:
    ▪ Oxymetazoline mixed with 4% lignocaine
    ▪ Adrenaline 0.25 ml 1:1 000 mixed with 20 ml 4% lignocaine
  » if source identified, use chemical (silver nitrate) ◇ or electrical cautery ◈
• 3rd line: packing:
  » Anterior nasal packing (after prep with vasoconstrictive/anaesthetic agent) (📖 p. 832)
  » Traditional ribbon gauze pack or commercial self-expanding or inflatable packs could be used

Posterior epistaxis (or if anterior techniques fail to control bleeding)

- Apply vasoconstrictive/anaesthetic agents
- Placement of posterior nasal packing – (often necessary to place bilaterally) (📖 p. 832)

Critical documentation

Record clinical findings, evidence of anaemia or shock, signs of trauma or a secondary cause, and location of bleed. Describe interventions (cautery, packing, and medications used) and response.

Disposition

Admit patients with persistent or significant bleeding, co-morbidities or those with posterior packs. Discharge other patients. Refer to ENT those with posterior bleeds, or anterior with complicating co-morbidities.

72 Otitis

Otitis is inflammation of the ear, usually infective in nature. Two broad categories exist: otitis media and otitis externa.

Otitis media is infection of the middle ear, particularly common in children. Common organisms are *S. pneumonia*, *H. influenzae*, *M. catarrhalis* and respiratory viruses. The cardinal symptom is ear pain; principal dangers are local invasion or systemic sepsis.

Otitis externa is inflammation and infection in the external auditory canal, sometimes involving the pinna. Common predisposing factors include minor trauma (over-zealous cleaning of the ears) and excessive water exposure (swimmers and surfers). Commonly caused by *S. aureus*, *Pseudomonas* (especially diabetics), HSV and occasionally fungi. Cardinal symptoms are ear pain and discharge; principal danger is local spread and suppuration.

History and physical examination

Key historical features

- Of the otitis: ear pain, discharge, decreased hearing, tinnitus, headache, fever
- Of local complications: headache, focal neurological deficits, cranial nerve deficits, AMS, vertigo, imbalance, ataxia, deafness, vomiting
- Of systemic complications: features of sepsis

Signs and symptoms

Otitis media

- Test hearing acuity, coordination and look for complications above
- Perform otoscopy:
  - Tympanic membrane (TM) may appear red and inflamed or dull
  - Visible middle ear effusion (bulging TM, air/fluid level behind TM, immobility of TM), poor visibility of middle ear structures
  - May have perforated TM with bleeding or purulent discharge

Otitis externa

- Redness and swelling of the external ear canal and sometimes pinna
- Discharge from the ear (may be offensive); also look for exostosis
- Pulling on the ear causes pain in the canal
- Look for signs of local or systemic complications as mentioned above
- ‘Malignant otitis externa’: otitis externa with osteomyelitis, chondritis and necrosis, usually *Pseudomonas* species, often in diabetics. Local destruction and systemic complications can progress rapidly and may lead to disability or death

Differential diagnosis

- Foreign body
- Tympanic membrane perforation
- Cerumen impaction
- Trauma
- Mastoiditis

Investigations

Clinical diagnosis. Consider swab with culture if resistant or recurrent. Consider CT ⟐ if concern about local suppuration or intracranial spread.

Management

The goal of acute management is to identify and manage dangerous complications, and provide symptomatic and specific treatment.

Otitis media

- Analgesia and antipyretics as needed
- Most cases can be managed expectantly without antibiotics
- Consider antibiotics for:
  - » Ill-appearing patients and those with local or systemic complications
  - » Children < 6 months old; older children if ill or certain diagnosis
  - » Immunocompromised patients
- Oral antibiotics for 7–10 days: amoxicillin 45 mg/kg BD is standard
- Penicillin allergy: cephalosporins, clarithromycin, or co-trimoxazole ◇
- Antihistamines, steroids and decongestants add no benefit
- In chronic or recurrent otitis media with effusion, antibiotics may not help; patient may need referral to ENT for myringotomy tube placement

Otitis externa

- Good aural toilet is key: gently clean external ear canal with cotton tipped wire
- Consider placing a cotton wick to promote drainage despite canal oedema
- Topical treatment with topical drops is usually sufficient:
  - » Acetic acid with or without steroids
  - » Hydrocortisone, neomycin sulphate and polymyxin B, ciprofloxacin or ofloxacin
- IV antibiotics for diabetics, or severe cases

Critical documentation

Note clinical findings, diagnosis, management plan.

Disposition

Admit patients with local complications or systemic sepsis. Refer to ENT if you cannot examine the ear due to gross swelling or if you suspect a dangerous alternative diagnosis or complication. Admit all diabetics with otitis externa for IV antibiotics.

73 ENT foreign bodies

ENT foreign bodies are common, especially in children. Presentations range from trivial to life-threatening (airway foreign bodies). Always consider foreign bodies in the differential diagnosis for any ENT or respiratory complaint.

The first five minutes

ABCs, particularly for respiratory complaints.

History, physical examination and differential diagnosis

See Table 73.1.

Investigations

- For ear and nose, clinical diagnosis.
- For foreign bodies in the throat which are not detected on examination (including indirect laryngoscopy):
  - » Imaging: AP and lateral neck XR/CXR (radio-opaque objects; if non-radioopaque – may see air contrast, compared to the surrounding soft tissue; bronchial FB – hyperinflation on affected side, may see a post obstructive pneumonia) ◇
  - » Bronchoscopy (for any suspected tracheal/bronchial FB) ⟐

Management

The goal of acute management is identification and removal of the foreign body. See Table 73.1.

Critical documentation

Record clinical findings, suspected foreign body and location, how removed, and what was removed.

Disposition

- Ear and nose: discharge; ENT referral for complicated cases
- Throat: discharge if easily dislodged; ENT referral if suspected retained/aspirated

Table 73.1 History, presentation, physical examination, differential diagnosis and management of ENT foreign bodies

	History	Clinical presentation	Physical examination	Differential diagnosis	Management
Ear	**Adults** 'Something is in my ear' Hearing deficit **Children** Painful ear/tinnitus Tugging at ear Bleeding Otorrhoea Hearing deficit	History of previous symptoms or procedures Severity and onset of pain Severe ear pain from movement or a sound in the ear – suspect live insect Associated symptoms (feeling movement, vertigo, gait disturbance)	**Adults** Communicate clearly Otoscope exam – large speculum **Children** Consider sedation **All** Retract pinna in posterior-superior direction using an otoscope Size, shape, composition? TM perforation	Otitis media Otitis externa Wax impaction Tympanic membrane perforation Cholesteatoma Auditory canal trauma Insect in ear	• Insect – 2% lignocaine or mineral oil to kill and facilitate removal • Irrigation with water at body temperature • Suction can be used for smooth objects (use controllable low pressure suction) • Other techniques – using alligator forceps under direct visualisation, or cyanoacrylate glue (superglue) on an applicator (e.g. paperclip or swab stick) – do not glue the applicator to the patient's ear canal!
Nose	**Adults** Insect/parasite in chronically ill, diabetics **Children (preschoolers)** Food, beads, toys Disk batteries (cause significant morbidity) Unilateral URI symptoms with purulent drainage	Vague c/o unilateral obstruction, epistaxis, sneezing, pain, fever Facial cellulitis	Need excellent light and nasal speculum Obvious FB in nares Foul exudate from a single nostril Visualise Examine bilateral nares	Idiopathic epistaxis Sinusitis Tumour Nasal polyps	• Prepare affected nasal cavity with topical vasoconstrictor/anaesthetic. » Adrenaline 0.25 ml of 1:1 000 mixed with 20 ml 4% lignocaine • Blow nose with unaffected nostril occluded • In young children, have parents blow into patient's mouth with unaffected nostril occluded (called 'parent's kiss' method) • Long alligator forceps or right angle hooks • Pass Foley catheter past FB, then inflate balloon and withdraw • Suction can also be attempted • Must always have concern for displacement of FBs in the airway

Throat/ airway	**Adults** Usually older, often with existing neurologic, GI, or ENT problems Adults can localise position: Mandibular angle – pharynx; lateral to trachea to clavicle – cervical oesophagus; substernal pain – oesophageal (aortic arch to lower sphincter) Acute oesophageal impactions typically present in first 12 hours **Children** Present late Can have vague history	Odynophagia – pharyngeal FB Dysphagia – lower oesophageal FB Retained FB: otalgia, cough, hoarseness, progressive lateralised pain Paediatric: can have subtle to severe symptoms Paediatric: tracheal compression from oesophageal FB	Apnoea = complete obstruction at pharyngeal, laryngeal or tracheal area Airway compromise: partial – cough, choking, dyspnoea Stridor = laryngotracheal FB Wheeze/rectractions - bronchial FB Drooling, inability to eat – lower oesophageal FB	Pharyngeal FB Laryngeal FB Oesophogeal FB Tracheobroncial FB Croup Epiglotitis Reactive airway disease Neck mass Pneumonitis Residual oropharyngeal trauma after spontaneously resolved obstruction Infection/abscess	(see Adult resuscitation p. 8 and Paediatric resuscitation p. 18) for more details) Heimlich manoeuvre, stomach-thrusts, and back-blows (as appropriate for age) for acute aspiration Apnoea = complete obstruction with need for immediate airway manoeuvres Jaw thrust, emergency laryngoscopy if not able to dislodge FB, use a bougie to push into the right main stem Oxygenate via emergency cricothyroidotomy (needle cricothyroidotomy in < 12 years).

74 Head and neck masses in adults

A neck mass is a relatively common presenting complaint. Many are innocent and self-limiting, but a neck mass may sometimes herald a serious underlying problem. The neck contains a very high density of structures and organs; abnormalities of any may produce a neck mass.

Most neck masses are caused by:

- Infection/inflammation (including reactive lymphadenopathy)
- Tumours (benign and malignant, including metastatic disease)
- Congenital masses, skin abnormalities and enlarged structures or organs of the neck (cysts, enlarged thyroid etc.)

The first five minutes

Usually not life threatening, but check for airway compromise and treat as needed.

History and physical examination

Knowledge of normal anatomy of the head and neck is essential.

Key historical features

Ask about:

- The mass – when was it noticed, how fast has it grown, previous episodes
- Associated features – pain, dysphagia, voice change, skin infections, weight loss, constitutional symptoms etc.
- The patient – pre-existing medical conditions (particularly malignancy and TB), medication and allergy

Important **'red flag' features** include:

- Age > 45
- Smoking and alcohol use
- HIV, TB or malignancy
- Change in voice or dysphagia
- Weight loss
- Systemic illness

Signs and symptoms

- A complete examination of the mass and surrounding structures, using an anatomical approach, is essential. Note any airway or vascular compromise
- Examine and describe the mass (size, location, relationship with surrounding structures, hard vs. soft/cystic, tenderness, skin changes, mobile vs. fixed, draining sinuses, any associated abnormalities of surrounding structures)
- Internal examination of the mouth, nose and throat
- Consider endoscopic examination of the pharynx in all cases presenting with red flag features or if concerned about underlying malignancy

Differential diagnoses and possible causes

Classification:

- Congenital abnormalities:
  - » Lateral neck masses (cysts, sinuses, fistulae)
  - » Central neck masses (thyroglossal cysts)
- Inflammatory (lymphadenopathy – self-limiting over weeks usually)
- Infective (bacterial, viral, mycobacteria, HIV, cat scratch)
- Trauma (haematoma, pseudoaneurysm)
- Metabolic, idiopathic, autoimmune (gout, sarcoid)
- Neoplasm:
  - » Benign (lipoma, haemangioma, cystic hygroma)
  - » Malignant (primary, or metastasis)

Investigations

Investigation is guided by the suspected cause and patient context. In patients with a clear URTI and reactive lymphadenopathy, an expectant approach is appropriate. In those with red flag features full evaluation, including endoscopy and biopsy may be needed. Tailor the investigations to the scenario. Always consider TB and HIV associated lymphadenopathy in areas of high disease prevalence.

Management

The goal of acute management is to identify and treat the small subset of patients with emergency features (airway compromise). If absent, thorough evaluation and management directed at the cause is needed.

Critical documentation

Note clinical and special investigation findings, diagnosis and management plan.

Disposition

Most patients can be followed up as outpatients. Refer patients with red flags to a specialist service as soon as possible.

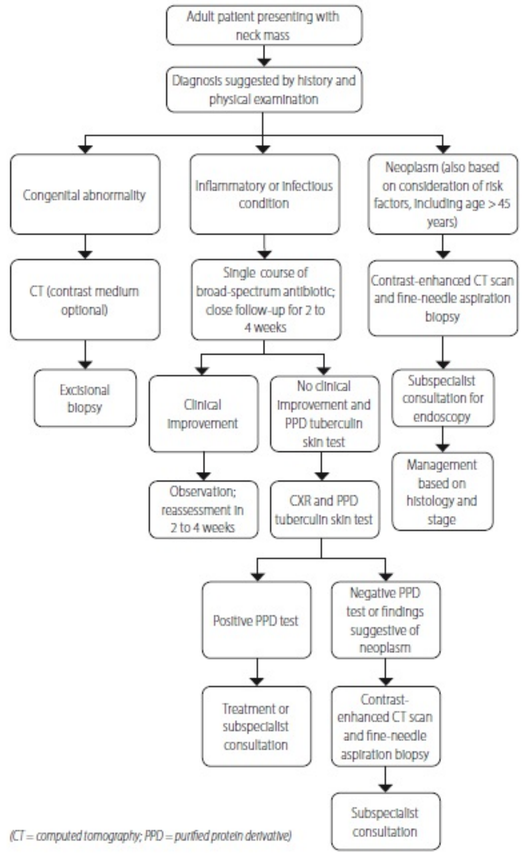


Figure 74.1 Evaluation and management of a neck mass in the adult patient

75 Head and neck masses in children

Head and neck masses are common; 90% are benign. Classification can be based on pathology: infectious, cystic, and benign or malignant neoplasms.

The first five minutes

Usually not life threatening; check for airway compromise and treat as needed.

History and physical examination

Key historical features

Varies:

- Infectious: fevers, recent upper respiratory or GI illness, contacts, exposure to cats, ingestion of uncooked meat
- Cystic: often present from birth, fluctuating size
- Neoplastic: insidious onset, constitutional symptoms

Signs and symptoms

If torticollis, trismus, dysphagia or odynophagia is present, suspect deep space infection.

- Infectious:
  - » Viral: small, rubbery, mild tenderness, usually bilateral
  - » Bacterial: larger 2–6 cm, tender, surrounding erythema, usually unilateral

» Granulomatous: insidious and chronic, submandibular or preauricular, spontaneous drainage
• Cystic: soft, ballotable, mobile, can move with swallowing, often midline (thyro-glossal duct and dermoid cysts) or anterior triangle (branchial apparatus cyst)
• Neoplastic: hard, non-tender:
  » Suspect malignancy if mass is immobile, fixed to underlying structures, or located in the posterior triangle. However, lymphoma is mobile and commonly in the anterior triangle

Possible causes and differential diagnosis

Table 75.1 Possible causes and differential diagnosis of neck mass in children

Infectious	Cystic	Neoplastic
Cervical lymphadenitis	Congenital malformations	Benign lesions
Suppurative lymph nodes	Thyroglossal duct cyst	Fibromatosis colli
Granulomatous disease	Branchial apparatus cyst	Haemangioma
Mycobacterium species	Fistulas	Goiter
Fungal infection	Dermoid cysts	Malignant lesions
Cat scratch disease	Lymphatic malformations	Lymphoma
	Lymphangioma	Rhabdomyosarcoma
		Neuroblastoma
		Metastatic adenopathy

Investigations

Guided by the suspected cause and patient context. In patients with a clear URTI and reactive LAN, an expectant approach is appropriate. In those with red flag features full evaluation, including endoscopy and biopsy may be needed. Tailor the investigations to the scenario. Always consider TB- and HIV-associated LAN in areas of high disease prevalence.

Management

The goal of acute management is to identify and treat the small subset of patients with emergency features (airway compromise). If absent, thorough evaluation and management is directed at the cause.
• Cervical lymphadenopathy: ≤ 1 cm is normal – no treatment necessary
• Infectious:
  » Cervical lymphadenitis: most commonly viral with no specific treatment. If bacterial infection is suspected treat with amoxicillin/clavulanic acid or clindamycin to cover *S. aureus* and *S. pyogenes*
  » Granulomatous and fungal lymphadenitis: treat with surgical excision
  » Cat scratch disease: treat with heat and analgesics
• Cystic:
  » Congenital and lymphatic malformations: treat with surgical excision. Make sure to rule out and treat a secondary infection
• Neoplasm:
  » Benign:
    ▪ Fibromatosis colli: spontaneous resolution in eight months with gentle stretching or physical therapy
    ▪ Haemangioma: spontaneous resolution over 10 years or surgical excision if airway compromise
    ▪ Goitre: preform thyroid function tests; consider surgical management
• Malignant masses:
  » Refer for chemotherapy and radiation

Critical documentation

Document airway patency and presence/ absence of systemic signs of illness; note diagnosis and management plan.

Disposition

Discharge most patients; refer for surgical or oncology management for cystic or neoplastic masses.

76 Sinusitis

Sinusitis is infection of the paranasal sinuses; it is relatively common and the cardinal feature is facial pain. It may be classified by duration of illness (acute < 4 weeks; subacute: 4–12 weeks; chronic: > 12 weeks) or by pathogen (virus – most common (rhinovirus, influenza, parainfluenza); bacteria – < 2% of cases (*S. pneumoniae, H. influenzae, M. catarrhalis, S. aureus*); fungal – rare – in immunocompromised patients (*Mucomycosis, Aspergillus*)).

The first five minutes

Almost never present an acute threat to life. Resuscitate as needed.

History and physical examination

It is very difficult to determine the aetiology of sinusitis (viral vs. bacterial) based purely on clinical presentation. Uncomplicated sinusitis of all causes will typically resolve spontaneously in 7–10 days.

Key historical features

Ask about prior history of sinusitis, immunocompromise.

Signs and symptoms

- Facial pain and pressure which typically worsens when bending forward
- Fever
- Nasal congestion; discharge (may be bloody or purulent)
- Headache
- Tooth pain occurs when infection extends into maxillary dental roots
- Anosmia (inability to smell) or hyposmia (decreased ability to smell)

Nasal examination

- Mucosal oedema, rhinorrhoea, discharge
- Polyps or septal deviation may predispose a patient to bacterial sinusitis

Facial examination

- Palpation of the sinuses may elicit pain. More likely bacterial sinusitis if unilateral

Eye, neurological and dental examination

- Normal in uncomplicated sinusitis
- If abnormal findings, consider serious complication or other diagnosis

Complications are rare, but occur more frequently in the immunocompromised and at the extremes of age:

- Intracranial: meningitis, epidural abscess, venous sinus thrombosis, brain abscess
- Extension into orbit: orbital cellulitis, periorbital cellulitis, orbital abscess
- Extension to surrounding bone: facial osteomyelitis, Pott’s puffy tumour (periosteal abscess of frontal bone)
- Systemic sepsis

Differential diagnosis

Includes allergic rhinitis, dental infections or caries, facial cellulitis or abscess, and foreign body in the nose.

Investigations

A clinical diagnosis. No testing is needed. XR of the sinuses does not distinguish between aetiologies. If complications are suspected, CT may help ⟐.

Treatment

The goal of acute management is to reduce pain and limit complications.

Antibiotics

Acute bacterial sinusitis will resolve without antibiotics in 75% of patients.

Criteria (if suspected acute bacterial sinusitis)

- **Persistent**: > 7–10 days of symptoms
- **Severe**: temp > 39°C for > 3 days, or purulent rhinorrhea with facial pain for > 3 days
- **Worsening** symptoms after initial improvement
- Immunocompromised patients should be treated with antibiotics

Agents (all for 10–14 days)

- Amoxicillin/clavulanic acid: first line. 500/125 mg po TID
- Doxycycline: 100 mg po BID. Not recommended for use in children
- Amoxicillin: 500 mg BID (high levels of resistance in some communities) 5–7 days

Pain relief with simple analgesics; saline irrigation (rinsing nasal passages with buffered sterile saline solution may decrease severity of symptoms; topical glucocorticoids (not recommended) large doses reduce duration of illness by 1–2 days; decongestants should be used sparingly; oral decongestants can be helpful for patients who have eustachian tube dysfunction (ear fullness, ear popping, decreased hearing).

Critical documentation

Record risk factors for complications and severe infections; eye and neurological examination.

Disposition

Admit patients with complications. Discharge all other patients.

77 Salivary gland problems

Diseases of the salivary glands are relatively rare, and are not usually acutely dangerous. The most common condition in childhood is viral parotitis. Always consider malignancy, especially in adults and with unilateral involvement. Consider salivary gland or duct stones, cysts, infections and abscesses.

The first five minutes

These conditions almost never present an acute threat to life.

History and physical examination

Cardinal symptoms: pain, swelling, fever, dryness of the mouth or altered taste.

Sialolithiasis

Calcium-rich stones, related to:

- Dehydration
- Decreased food intake (lowers demand for saliva)
- Medications that decrease saliva production (antihistamines, anti-hypertensives and psychiatric medication)
- May be asymptomatic or painful with swelling and dry mouth

Sialadenitis

A painful infection; usually bacterial. More common among elderly with salivary gland stones. Can also occur during the first few weeks of life.

Viral infections

Presentation: facial swelling, pain, fever and difficulty eating. Common viruses: mumps, infectious mononucleosis. Rare complications: orchitis, pancreatitis.

Cysts

May be idiopathic or secondary to traumatic injuries, infections, or salivary gland stones or tumours.

Benign tumours

Most salivary gland tumours occur in the parotid gland. The majority are benign and present as slow-growing, painless masses at the angle of the mandible. Risk factors include radiation exposure and smoking.

Malignant tumours

Rare. Risk factors include smoking, radiation exposure, and Sjögren's syndrome (a chronic autoimmune disorder; typically middle aged women; in about 50%, it occurs together with rheumatoid arthritis, SLE, scleroderma or polymyositis).

Investigation

Investigations should be guided by the clinical scenario.

- Imaging: facial XR ◇ (may detect stones); CT face (detect tumours and stones);sialography (detect duct blockage) ⟐

Management

The goal of acute management is relief of symptoms and identification of potentially dangerous conditions. Treatment varies according to the disorder.

Sialolithiasis

- Superficial stones – direct manipulation
- Deep stones – surgery
- Warm compresses on the infected gland
- Encouraging saliva flow (chew sour, sugarless candies or drink orange or lemon juice)
- Occasionally, antibiotics or incision and drainage may be required

Viral infections

Treatment is symptomatic.

Cysts

Spontaneous drainage may occur with smaller cysts. Larger cysts need surgery.

Benign tumours

Surgical excision.

Malignant tumours

Need surgery, radiation or chemotherapy. Refer early.

Sjögren's syndrome

Treatment is directed at the main symptom – dry mouth. Good oral hygiene should be combined with medication (to stimulate more saliva secretion (pilocarpine and cevimeline). Sugarless gum and candy can stimulate saliva production. Avoid medications that make dry mouth worse.

Critical documentation

Note clinical findings, investigation results, diagnosis, management plan.

Disposition

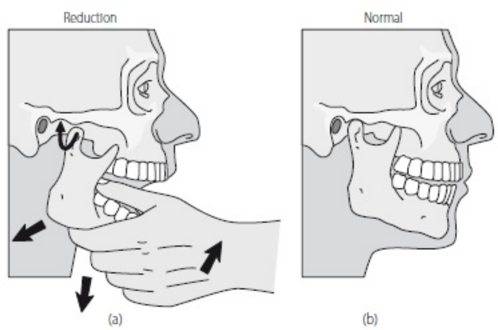
Refer to ENT if there is concern of malignancy. Discharge most patients.

78 Temporomandibular joint dislocation

Temporomandibular joint (TMJ) dislocation is a painful condition whereby the mandibular condyle dislocates from the temporal bone and becomes fixed in the antero-superior aspect of the articular eminence (Figure 78.1).

Usually the dislocation is anterior but may also rarely be posterior or superior, especially when associated with facial or skull trauma.

Figure 78.1 A dislocated (a) and a normal (b) mandible



The first five minutes

Usually an isolated injury. Look for associated serious injuries and resuscitate as needed.

History and physical examination

The clinical presentation is typical: the patient presents with an inability to close the mouth and pain around the TMJ. The dislocation may be unilateral or bilateral. It may have been the result of direct trauma but is often associated with wide opening of the mouth, such as yawning.

Ask about previous similar episodes, jaw trauma or surgery. Remember to look for evidence of spinal or other associated injuries.

Differential diagnosis

- The diagnosis is usually obvious
- If associated with severe facial swelling secondary to trauma diagnosis may be more difficult and XR or CT scan may be required

Management

The goal of acute management is reduction of the dislocation.

- Simple TMJ dislocations (unilateral or bilateral):
  - » Manual reduction preferred; may sometimes be achieved without any analgesia, especially in patients with

recurrent dislocation

» Consider analgesia or procedural sedation

» **Intra oral technique**: provider places their gloved thumbs (wrap in gauze to protect thumbs) in the mouth of the patient and firmly pushes downward on the molars while rotating the chin upwards. The motion is downward pressure while at the same time lifting the chin to reduce the condyle (Figure 78.1 (a) shows the direction of movement to reduce)

- **Extra oral technique**: gentle firm consistent pressure over the visible protruding condyles may be effective in reducing the dislocated condyles
- Complicated/recurrent dislocations with or without associated fractures may require reduction in theatre:

» After reduction, soft diet and jaw support when opening the mouth wide for 1–2 weeks

Figure 78.2 Reduction of dislocated temporomandibular joint

Critical documentation

Note mechanism, other injuries found or excluded, reduction method, pharmacology used.

Disposition

Discharge once the TMJ is reduced, ENT follow-up is suggested, especially if complicated or recurrent. Refer all patients with fracture-dislocation to a facial surgeon.

References

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Part

4

E. Environmental emergencies

79 High altitude sickness

High altitude sickness (HAS) encompasses pulmonary and cerebral oedema from uneven vasoconstriction and capillary leak, triggered by exposure to hypobaric hypoxia. HAS includes acute mountain sickness, high altitude cerebral oedema (HACE) and high altitude pulmonary oedema (HAPE). These are related to rate and height of ascent, and typically occur at > 2 500 m.

The first five minutes

ABC, VS, O_2 (if available), plan descent.

History and physical examination

Key historical features

- Ascent profile/timing, prior HAS
- Symptom onset and progression

Signs and symptoms

- VS: tachycardia, tachypnoea, hypoxia
- Cyanosis, confusion, ataxia, crepitations, wheeze
- Lake Louise criteria:
  - » Acute mountain sickness: altitude ascent, headache and at least one of:
    - General: such as fatigue or weakness

- GI: nausea, vomiting, loss of appetite
- Neuro: dizziness, lightheadedness, insomnia, visual scotomata

» HACE: ascent plus:
- Altered mental status OR ataxia in a patient WITH acute mountain sickness
- Altered mental status AND ataxia in a patient WITHOUT acute mountain sickness

» HAPE: ascent with:
- At least two of the following *symptoms*:
  - Dyspnoea at rest, cough
  - Decreased exercise tolerance or weakness
  - Chest tightness or congestion

AND
- At least two of the following *signs*:
  - Crackles or wheezing in at least one lung field
  - Central cyanosis, tachypnoea, tachycardia

Possible causes and differential diagnosis

- High altitude headache
- Hypothermia, hypoglycaemia, hyponatraemia
- Dehydration, carbon monoxide toxicity, stroke
- Pulmonary embolism, pneumonia

Investigations

- Imaging: CXR ◇ if available (if hypoxic or abnormal pulmonary exam)

Management

The goal of acute management is to ensure oxygenation and halt progressive pathology by descent.

Acute mountain sickness/HACE

Prevention:

- Gradual ascent
- Acetazolamide 125 mg PO twice daily
- Dexamethasone 2 mg PO every 6 hours or 4 mg PO every 12 hours

Treatment:

- Descend until symptoms resolve
- Portable hyperbaric chamber (if descent is not possible) ◈
- Oxygen to maintain SaO_2 90% (if available) ◇
- Acetazolamide: 250 mg PO every 12 hours
- Dexamethasone: 8 mg IV/IM/PO once then 4 mg IV/IM/PO every 6 hours

HAPE

Prevention:

- Gradual ascent
- Drug prophylaxis if prior history of HAPE
- Nifedipine 60 mg extended release PO daily
- Salmeterol 125 mcg INH inhalation BID
- Acetazolamide 125 mg PO BID

Treatment:

- Immediate descent
- O_2 to maintain SaO_2 90% (if available) ◇
- Portable hyperbaric chamber (if descent is not possible) ◇
- Nifedipine 60 mg ER daily (minimal research supporting this)

HAPE plus AMS/HACE

- Immediate descent
- HAPE treatment plus dexamethasone
- Use caution when adding vasodilators due to risk of dropping BP

Critical documentation

Ascent profile and altitude when symptoms started. Record abnormal VS and serial exam findings. Document cardiac, pulmonary and neurological exams.

80 Heat-related illnesses

Heat-related disorders include *heat exhaustion* (collapse with near normal core temperature and intact sweating) and *heat stroke* (collapse with temperature > 40 °C ± sweating). Both are medical emergencies requiring immediate cooling.

The first five minutes

- ABC, VS, O_2, IV access, cardiac monitor ◇
- Differentiate heat exhaustion from:
  - » Heat stroke - obtain an accurate core temperature (rectal)
  - » Over-hydration with cerebral oedema (dilute urine) in athletes
- Start rapid cooling by tepid sponging and active fanning

History and physical examination

Key historical features

- Risk factors: extremes of age, dehydration, medications (e.g. anticholinergics)
- Recent illnesses, medication changes (including discontinuation) within last six weeks, family history, prior occurrence, living and work conditions (environmental exposure)
- Heat exhaustion:
  - » Generalised malaise, fatigue, headache, nausea, vomiting
  - » Core temperature may be elevated or normal (< 40 °C)
  - » No neurological symptoms
  - » Sweating present
- Heat stroke:
  - » Heat exhaustion symptoms
  - » Neurological symptoms: abnormal behaviours, irritability, AMS
  - » Higher core temperature (> 40 °C)
  - » Sweating may or may not be present (not a sensitive sign)

Signs and symptoms

- Tachycardia, tachypnoea, hyperventilating; normotensive with wide pulse pressure (hypotension may be present secondary to peripheral vasodilation or myocardial ischaemia)
- Neurological symptoms indicate heat stroke

Possible causes and differential diagnosis

Drug associated

- Toxicity: anticholinergic, salicylate, stimulants
- Malignant hyperthermia: associated with suxamethonium or halothane
- Serotonin syndrome
- Neuroleptic malignant syndrome: classic antipsychotic medications
- Drug/alcohol withdrawal

Neurologic

- Intracranial haemorrhage
- Status epilepticus
- Hypothalamic injury
- Encephalopathy

Other

Infection, thyrotoxicosis, pheochromocytoma, diabetic ketoacidosis (all unlikely).

Investigations

Clinical diagnosis. Consider:
- Labs: CBC, electrolytes, renal, glucose ◇; CK ◈
- ECG ◇: (dysrhythmias, nonspecific ST - T wave changes)
- Imaging: CXR ◇ (non-cardiogenic pulmonary oedema)
- Toxicology screen, CT head ◈ and LP if CNS aetiology suspected in hyperpyrexia

Management

The goal of acute management is to bring core temperature to < 40 °C in 30 minutes**.**

Rapid cooling

- Can be discontinued at temperature of 39 °C (rectally).
- Clothing removal and sponge baths/shower: may be enough for mild cases
- Immersion of the entire body (except head) in ice or cool water (1.7 °C to 14 °C), while keeping the water circulating ◇. Fastest method, but impractical for monitoring and resuscitation
- Evaporative cooling:
  - Fans blow warm air as the patient is sprayed with cooled atomised water (15 °C) ◇
  - Easier to implement and allows simultaneous care of the patient
  - Can be used for multiple patients at the same time
- Other adjuncts/temporising measures:
  - Ice-packing the neck, axillae and the groin. Avoid shivering!
  - Rotating ice/wet towels over the entire body every few minutes
  - Cooling blankets ◈
  - Cool humidified O_2, cool IV fluids, cold gastric/rectal lavage ◈
  - In extreme cases: cold peritoneal lavage ◈

Other therapies

Aggressive hydration (if fluid depleted): with IV fluids; watch for pulmonary oedema.
Benzodiazepines: to decrease shivering (and heat production).
Antipyretics: no role, may actually cause harm.

Critical documentation

• Mental status stroke vs exhaustion). Serial temperature Q30 min.

Disposition

Patients with heat exhaustion can be discharged after cooling and hydration if at baseline. Patients with heat stroke should be admitted to monitored unit ⟐.

81 Hypothermia

Defined as a core body temperature less than 35 °C; can occur in nearly any climate or weather. Aggressive rewarming and management of complications are vital.

The first five minutes

• ABC, VS, O_2, cardiac monitor, IVF
• Palpate central artery for up to one minute before initiating CPR
• Determine core body temperature
  » Oesophageal measurement is best, followed by rectal, oral, then axillary
  » Other sites will lag behind by > 0.5 °C as patient is being warmed.
• Expose and dry patient, then initiate rewarming therapy (Table 81.1).

History and physical examination

Evaluate for precipitating causes and complications (see Table 81.1).
• Water immersion or found down: water conducts heat away from the body at a rate of 25-times air, while stone floors conduct heat 100-times faster than air
• Comorbidities: extremes of age, substance abuse, malnutrition, sepsis, TB, HIV/AIDS, diabetes, cardiovascular or neurological disease, trauma, hypothyroidism, hypopituitarism
• Medications: sedative-hypnotics, opiates, antipsychotics, thyroid replacement, beta-blockers, insulin, oral hypoglycaemics, and traditional medications/herbals

Possible causes and differential diagnosis

Exposure is the most common cause of hypothermia. Many medical conditions can result in hypothermia (e.g. hypothyroidism, adrenal insufficiency, sepsis); medications directly or indirectly cause hypothermia.

Investigations

Core temperature in all (Table 81.1); other investigations by suspected cause or complications. ECG in all ◇.

Management

The goal of acute management is to raise core temperature safely (Table 81.1).
• Hyper/hypoglycaemia: renal and hepatic failure can lead to glucose derrangements; frequent glucose measurements
• Hypovolaemia: cold diuresis and insensible losses can lead to dehydration
• Acidosis: renal dysfunction, hypoperfusion, and bradypnoea can lead to combined respiratory and metabolic acidosis with elevated serum lactate

Critical documentation

Initial and serial core temperature, Q30 min, time of presentation and initiation of treatment, method of rewarming, possible/likely precipitating cause, time of repeat temperature and blood glucose.

Disposition

- Admit: moderate/severe hypothermia, extremes of age, or with multiple comorbidities
- ICU: patients who lost consciousness, required invasive rewarming, or experienced haemodynamic instability or cardiac arrest.
- Discharge: healthy, young patients with environmental exposures who are successfully rewarmed

Table 81.1 Stages, signs, and treatments for hypothermia

Core temp	35 °C–32 °C (Mild)		32 °C–28 °C (Moderate)		< 28 °C (Severe)			
Clinical features	Early: sympathetic excitation, hypertension, tachypnoea, tachycardia, pallor, shivering Late: confusion, hepatic and renal dysfunction, hyperglycaemia, apathy, loss of shivering response		Disorientation, slurred speech, loss of memory and manual dexterity, dysarthrias, bradycardia, miosis, bradypnoea, numbness, hypotension, hyporeflexia, J-Wave appears		Depressed cardiac conductivity, weak pulse, progressive decline in consciousness, apnoea, coma, oliguria, pulmonary oedema, nonreactive pupils			
Preferred method	Passive	Expose, dry, and wrap patient in warm, dry blankets	Active ◇	Expose, dry, then use skin-to-skin, forced air, or heat lamps	GI lavage ◇	Lavage with boluses of warm fluid (250 ml at 40 °C) via NG	By-pass/dialysis ◈	Emergency transfer as needed
Rate of warming (°C/hour)	2		1–3		1–2		4–9	
Ideal patient	Young or otherwise healthy patients		Paediatrics		Multiply comorbid individuals		Patients with cardiac arrest	

82 Radiation exposure

Radiation can involve contamination (the radioactive material is IN or ON the body, and the patient remains radioactive and a risk to contacts), or exposure (the patient suffers the effects but poses no threat to staff).

Radiation sickness occurs after exposure to more than 1 Gray (Gy). It usually begins within 48 hours but can appear up to six days later – there is a transient improvement in symptoms followed by reappearance of symptoms that usually last for weeks.

The first five minutes

ABC, VS, resuscitate as needed.

History and physical examination

Key historical features

Symptoms are dose dependent:

- Cutaneous: colour change, pruritus, blisters, hair loss, swelling
- Gastrointestinal: nausea, vomiting, diarrhoea, abdominal pain, bleeding
- Haematological: bleeding, easy bruising
- Neurological: fatigue, malaise, headache, confusion, fever, weakness, paraesthesia
- Pulmonary: shortness of breath

Signs and symptoms

- Cardiovascular: hypotension, severe dehydration, shock
- Cutaneous: erythema, pruritus, petechiae, blistering, ulceration, alopecia, oedema, desquamation, necrosis
- Gastrointestinal: abdominal pain, GI bleed, dehydration, ileus, bowel obstruction, electrolyte abnormalities
- Haematological: bleeding, pancytopenia (usually a late finding)
- Neurological: AMS, fever, cerebral oedema, cerebral anoxia, papilloedema, motor deficits, sensory deficits
- Pulmonary: dyspnoea, pneumonitis, acute lung injury, pulmonary oedema
- Renal: acute renal failure
- VS: febrile, tachycardia, hypotensive, tachypnoea, hypoxia

Possible causes and differential diagnosis

May be caused by industrial accidents or weaponised release. Differential includes many multi-system conditions.

Investigations

- Labs: CBC (baseline cell counts), electrolytes, renal function ◇
- ECG: ◇ (tachycardia, ischaemia, signs of electrolyte abnormalities)
- Imaging: CXR ◇ (pulmonary oedema, acute lung injury, acute respiratory distress syndrome, pneumonia); CT head ◈ (cerebral oedema, anoxia)

Management

The goal of acute management is supportive care and to limit exposure in cases of contamination.

Protect self-exposure by time (keep exposure as short as possible), distance (greatest possible distance from source), and shielding (appropriate clothing depending on source).

- Consider antibiotics, especially Gram negative coverage, if exposure $\geq$ 2 Gy
- Consider palliative care if exposure is $\geq$ 10 Gy
- Contact relevant reporting authorities

Critical documentation

- Radiation exposure: type and dose
- Symptoms
- VS, including temperature and pulse oximetry
- TBSA affected including per cent burn
- Full cardiac, pulmonary, GI, neurologic and cutaneous examinations
- Cell count nadirs

Disposition

Admit to ICU if exposure with severe symptoms or known exposure > 5 Gy ◈; admit for any symptoms and/or known exposure > 1 Gy.

83 Electrical and lightning injuries

Electrical injuries vary widely in patterns and presentations. Significant exposures can cause deep electro-thermal burns, internal damage, cardiac dysrhythmias and cardiopulmonary arrest. Victims are often young and healthy with significant potential for survival if immediate resuscitation is provided. Death is usually from immediate cardiopulmonary arrest.

The first five minutes

Rescue from the electrical source.

- ABC, VS, resuscitate as needed
- Immobilise cervical spine (victims may be thrown from source)

Possible causes

Extent of injury depends on amount and type of current conducted. Alternating current (AC) moves cyclically in two directions, while direct current (DC) moves in a single direction.

- AC is associated with tetanic muscle contraction that can force tight grip on the source. Extended exposures cause greater injury and risk of VFib
- DC is associated with higher voltages that can propel victims away from the point of contact and cause secondary injury from blunt trauma
- Lightning is a massive unidirectional impulse. Duration of current contact is typically short (1–2 milliseconds)

History and physical examination

Key historical features

Details of exposure (voltage, current, duration of contact and likelihood of secondary trauma). Duration of cardiopulmonary arrest and time to CPR.

Signs and symptoms

- Neurologic: confusion, LoC, seizure, cranial nerve deficits, paresis and keraunoparalysis (a transient paralysis) are common. Patients with prolonged paralysis are likely to have spinal-cord injury. Extremities may be initially pulseless, cyanotic, and weak (vasomotor instability)
- Secondary trauma: barotrauma (e.g. tympanic membrane rupture), orthopaedic injuries, traumatic brain and spinal cord injuries from falls
- Soft tissue: occult muscle injury may occur. Monitor for oedema, compartment syndrome
- Mucocutaneous: the hands are the typical site of contact and may show burns. Massive coagulation necrosis may underlie normal skin. Arc and splash burns are the most common heat-related injuries causing superficial and/or partial-thickness burns. Feathering burns or ‘Lichtenberg flowers’ are pathognomonic for lightning. Exposure to the mouth (i.e. children biting electric cords) may cause burns. Eschar formation near the labial artery is possible, with potential for significant haemorrhage if disturbed

Possible causes and differential diagnosis

Lightning, high voltage power lines, other thermal or chemical burns.

Investigations

- Labs: electrolytes, renal ◇; troponin, lactate, CK ◈
- ECG ◇
- Imaging: as indicated for trauma

Management

Prevention

Seeking safe shelter during lightning storms can be lifesaving.

- Count seconds from a lightning flash until thunder, then divide by 3 to estimate the storm’s distance in km
- 30/30 rule: most lightning occurs within 8–9 km of the previous flash. Take cover if the time from lightning flash to thunder is < 30 seconds. Stay in a safe place until no lightning or thunder for 30 minutes
- If outdoors, avoid ridge tops, open fields, or isolated tall poles/trees. Avoid moisture and conductive or electrical devices

Treatment

The goal of acute management is cardiopulmonary stabilisation and injury care.

- Standard treatment for arrhythmias

- Wound irrigation, debridement, and tetanus prophylaxis as necessary
- Maximise urine output with fluid resuscitation
- Frequent neurovascular exams to identify compartment syndrome
- Persistent adynamic ileus will require abdominal imaging or exploratory laparotomy to identify visceral injury

Critical documentation

Some findings are delayed or transient; frequent re-examination needed.

Disposition

Admit high-voltage injury. Transfer to burn centre when possible. Low-voltage injury and lightning strike survivors without neurological or cardiac insult can be discharged after observation.

84 Complications of smoke inhalation

The leading cause of death due to fires; causes three types of injuries – thermal and chemical injuries to the upper airways, pulmonary irritation, and asphyxiation, including by airborne monoxide and cyanide toxicity.

The first five minutes

ABC, VS, O_2, IV fluids, cardiac monitor ◇.

Intubate early if risk of oedema.

History and physical examination

Key historical features

Difficulty breathing, stridor, wheezing, rales, rhonchi, burns to the face, carbonaceous sputum. Details of event: closed space, explosion?

Signs and symptoms

Soot to the nose and mouth, oropharyngeal oedema.

- Carbon monoxide: if the patient is unconscious, assume CO and cyanide toxicity. If the patient has confusion, headache, vomiting, neurological symptoms, assume CO toxicity. The patient may have a falsely elevated or normal pulse oximetry. (📖 Cardiodepressant overdose p. 694)
- Cyanide toxicity: patient will have altered level of consciousness, dizziness, headache, tachycardia, tachypnoea, can have normal pulse oximetry readings (📖 p. 700)
- Methaemoglobinemia: anxiety, headache, dyspnoea, dizziness, coma, seizures (📖 p. 710)

Possible causes and differential diagnosis

- Thermal injury, hypoxic gas inhalation, chemical inhalation injury
- Consider hypoglycaemia, traumatic brain injury, seizures, and drug overdose

Investigations

- Labs: electrolytes, ABG ◇ (decreased tissue perfusion, oxyhaemoglobin saturation, carboxyhaemoglobin saturation, and methaemoglobin concentration), lactate (tissue hypoxia) ⟐
- Imaging: CXR ◇ (after 24 hours opacity may suggest ARDS, aspiration, or fluid overload); fiberoptic bronchoscopy ⟐ (confirms injury, clears debris)

Management

The goal of acute management is to protect the patient’s airway and provide supportive care.

100% FiO_2, early intubation (large lumen ET tube); bronchodilators (beta-agonists can help with bronchospasm); fluids.

See also toxicology chapters for management of methaemoglobinemia, carbon monoxide and cyanide exposure p. 710, p. 702 and p. 700.

Critical documentation

Document serial examinations of the airway including direct laryngoscopy.

Disposition

Consider transfer to a burn or critical care facility. If there is no upper airway injury, close observation for 24 hours is reasonable.

85 Complications associated with diving

Dive injuries can range from minor to life-threatening depending on dive profile, time, breathing gas and associated injuries. Immediate recognition, response with 100% O_2, relocation to a health facility and recompression therapy may be critical. Associated illnesses may include hypothermia, trauma, near-drowning, marine stings and bites, medical illness and dehydration.

The first five minutes

Rescue patient from open water. ABC, VS.

History and physical

Decompression sickness (DCS)

Clinical diagnosis. Occurs after ascent from a dive. Symptom onset within 24 hours in 99% of cases, 50% within an hour, with neurological symptoms occurring earlier in short deep dives. Most recreational injuries involve spinal decompression sickness.

- Type I: musculoskeletal pain, pain in larger joints (shoulder, elbow, knee), lymphoedema, rash, pruritis
- Type II: neurologic- central or peripheral, girdle pain radiating from the back, paraesthesia in legs, lower limb weakness, incontinence, cardio-respiratory symptoms

Barotrauma

- Pulmonary barotrauma
  - Arterial gas embolism (AGE): immediate AMS after ascent, convulsions, LoC, hemiplegia, hemiparesis or chest pain
  - Pneumomediastinum
  - Subcutaneous emphysema
  - Tension PTX: severe dyspnoea and cyanosis, tracheal deviation
  - Pneumoperitoneum
- Other barotrauma
  - Middle ear – ear pain, headache, tympanic membrane perforation and haemorrhage, deafness
  - Sinus – pain over frontal or maxillary sinus, epistaxis, blood in mask
  - Inner ear – vertigo, tinnitus
  - Mask – petechial haemorrhages on face, sub-conjunctival haemorrhage

Hypothermia

Core body temperature below 35 °C, shivering, muscle cramps, memory loss, delirium, drowsiness, muscle rigidity, progressive coma.

Near drowning

Dyspnoea, cyanosis, unconsciousness.

Differential diagnosis

Trauma, hypothermia, near drowning, contaminated diving gas, O_2 toxicity, carbon dioxide toxicity, carbon monoxide toxicity, immersion pulmonary oedema, water aspiration, stroke, myocardial infraction.

Investigations

- Labs: ABG, chemistry, CBC ◇, lactate ◈
- Imaging: CXR (PTX); upright AXR (perforated bowel) ◇; CT head/face (evaluate for AGE stroke and sinus rupture (pneumocephalus)) ◈

Management

The goal of acute management is to reverse tissue ischaemia by immediate high concentration O_2 therapy and earliest possible access to recompression for severe cases.

Decompression illness: 100% O_2, hydration, transfer to health facility (resuscitation) and then hyperbaric oxygen (HBO) facility for recompression ◈.

Hypothermia: removal from open water and rewarming measures.

Barotrauma: needle and then tube thoracostomy for tension PTX. Needle decompression of tension pneumoperitoneum. ◇. Recompression in HBO chamber for AGE ◈.

Near drowning: rapid rescue from open water, medical resuscitation with 100% O_2, monitor for delayed aspiration pneumonitis (CXR at 4–6 hours).

Ear and sinus ailments

- Middle ear barotrauma: no treatment or decongestants. If purulent ottorhoea give antibiotics, analgesia, and consult otolaryngology
- Inner ear barotrauma: bed rest, head of the bed at 30 degrees, avoid increased intracranial pressure. HBO therapy ◈
- Sinus, tooth, and facial barotrauma: decongestants and analgesia, otolaryngology consult for ruptured sinus ◇

Critical documentation

Time of symptom onset, dive information: log, breathing apparatus, dive profile, missed safety stops, activity prior to dive, detailed neurological exam.

Disposition

Call Divers Alert Network (DAN) 0800-020-111 (within Africa) +27-(0)-828 10 60 10 (accepts collect calls) or +1-919-684-9111.

Transfer to health facility and consult HBO Centre. May require ICU.

86 Drowning

According to the Utstein guidelines, drowning may be defined as ‘a process resulting in primary respiratory impairment from submersion/immersion in a liquid medium’. It is the third leading cause of injury-related death worldwide. Treatment is mainly supportive; prevention is paramount.

The first five minutes

ABC, VS, IV access, O_2, cardiac monitor, blood glucose, rewarming.

Use C-spine precautions if potential for trauma (or unwitnessed).

History and physical examination

Key historical features

- Location of drowning, length of submersion, temperature of body of water
- Preceding events, such as chest pain, seizure, syncope, trauma, or substance use/intoxication

Signs and symptoms

- Hypoxia, hypothermia, bradycardia, hypotension, arrhythmia
- Central and/or peripheral cyanosis
- Lung examination may reveal diminished sounds, rhonchi or crepitations

Possible causes and differential diagnosis

- Intoxication, seizure, syncope, cardiac dysrhythmia
- Acute respiratory distress syndrome (ARDS), hypothermia, head and neck trauma, (especially if a diving injury or fall from height)

Investigations

- Labs: CBC, electrolytes, ABG ◇
- ECG ◇: (dysrhythmias, ischaemia as possible cause)
- Imaging: CXR ◇ (pulmonary infiltrates, aspiration); C-spine XR ◇; CT spine, CT brain ◈ (if concern about trauma)

Management

The goal of acute management is to secure the airway (maintaining cervical spine precautions), restore oxygenation, and identify occult traumatic or medical causes.

Drowning patients with respiratory compromise or altered mental status

- Assisted respirations, crystalloid fluid resuscitation, blankets for rewarming
- O_2, warm IV fluids ◇
- Endotracheal intubation, active rewarming with heat lamps ◈

Drowning patients who are now asymptomatic

- O_2, blood glucose, six hour observation.

Critical documentation

Length of submersion, neurological examination, lung examination, response to O_2, temperature at start and end of resuscitation. Respiratory status at six hours.

Disposition

Drowning patients with respiratory compromise, altered mental status or intubated patients

Admit, preferably to ICU.

Drowning patients who are asymptomatic

Discharge if no hypoxia or other complaints after a 6–8 hour observation period. Instruct patients to return if they

develop symptoms (as there are reports of ‘secondary drowning’ in patients who initially appeared well).

87 Marine stings

There are several major causes of marine stings, sticks or bites including cnidarian (jellyfish, anemones, corals, hydroids, bluebottles), fish (catfish, stonefish, scorpionfish, lionfish, stingrays and weeverfish), sea urchins, sea snakes and octopi.

The first five minutes

ABC, VS, O_2. Hot water application to the sting.

History and physical examination

Key historical features

Patients can often report the source of sting. If the cause is unknown, all marine stings except sea snake and blue-ring octopus produce immediate pain.

Signs and symptoms

Jellyfish often leave tentacle marking(s) with erythema, vesicles or ecchymosis. These are commonly on extremities, but rarely on the palms and soles.

Anemones can cause an urticarial rash

Coral fish and urchin stings are frequently on hands and feet; sea snails often on the hand (from being held) and stingrays on the lower extremity (when stepped on).

With coral fish and urchins, examination may show lacerations or puncture wounds with retained foreign body.

Investigations

XR may be helpful if retained foreign body is suspected, but is not necessary for management in the emergency setting.

- Imaging: thoracic or abdominal penetrating wound, stingray ◇, CT, endoscopy or bronchoscopy ◈ (prior to removal of the spine if possible)

Management

Envenomation by marine animals is treated by hot water immersion (43–45 °C).

If visible, foreign bodies should be removed, however exploration for urchin spines is usually fruitless. Spines will be likely absorbed in days to three weeks, or work their way to skin for removal.

Analgesia

- Pain management is crucial for all stings
- Jellyfish and stonefish – hot water immersion may be sufficient (superior to cooling, baking soda or lidocaine)
- Urchin venom may also be denatured by 43–45 °C water

> NB: Several commonly reported methods for jellyfish sting treatment – fresh water, urine, ammonia, ethanol, meat tenderiser and methylated spirits – have been shown to potentially increase nematocyst firing, which may worsen symptoms and should be avoided.

Local wound care

Copious irrigation with salt water (fresh water activates nematocysts), removal of foreign body, tetanus as needed,

leave wound open.

Antibiotics

- Superficial wounds: none
- Puncture wounds: first generation cephalosporin (or clindamycin plus levofloxacin for MRSA and penicillin allergic patients) plus doxycycline if a seawater exposure

Antivenom (if available)

- Immediately for box jellyfish and sea snake
- In severe stonefish envenomation

Critical documentation

Aetiology of the sting and time from sting. Appearance and progression of lesions.

Disposition

Admit patients with severe complications, including cardiopulmonary involvement, preferably to ICU. If the cause is known and the patient is well, discharge with close (1–2 days) follow up (high risk of infection). Those receiving antivenin must remain in observation.

88 Hymenoptera (bees, wasps and ants)

Arthropod bites and stings are a problem worldwide and can cause mild to severe reactions, ranging from local irritation, to systemic toxicity and frank anaphylaxis. Hymenoptera (bees, wasps and ants) are responsible for the largest number of severe allergic reactions. The number of human deaths caused by hymenoptera exceed the number caused by all other venomous animals combined. Rapid onset reactions may become severe and can lead to death – 50% in 30–60 minutes, 75% within four hours. Early recognition of the type of insect is not essential.

The first five minutes

- Observe for signs of systemic toxicity and anaphylaxis. Remove stinger (by scraping or flicking, do not pinch). Apply cold compresses to area of sting and irritation; elevate limb
- ABC, VS, facemask O_2, IV access, cardiac monitor ◇
- Adrenaline (1:1 000) 0.3–0.5 mg IM, every 3–5 minutes as needed for signs of anaphylaxis

History and physical examination

Key historical features

- Inquire about previous insect bites and the reaction to them.
- Timing of exposure, number of stings

Signs and symptoms

- Wound site may show stinger, surrounding local skin inflammatory reaction, bleeding with oedema (bees), or papules (fire ants)
- Severe reaction: nausea/vomiting, facial swelling, tachypnoea/brochospasm, hypotension, dizziness, chest/abdominal pain
- Abdominal cramps – mimics peritonitis. Chest pain – mimics angina.

Possible causes and differential diagnosis

Cellulitis, snake bite, cat scratch disease, angina, urticarial vasculitis, asthma.

Investigations

Labs: investigations are usually only needed in seriously ill patients.

Management

The goal of acute management is to relieve symptoms, and address allergic reactions and systemic toxicity.

Mild reactions to bee and ant stings

- Remove stinger and wash sting site with soap and water, apply intermittent cold compresses, consider topical benzocaine 20%/menthol 0.5% ointment, 1% hydrocortisone for one week if extensive local inflammation
- Elevate affected area if significant swelling
- Antibiotic ointment for ant stings as well as the above treatment. For rash/fever, use cephalexin for one week. Do not deroof vesicles

Massive/multiple stings

- Prepare to treat anaphylactic reaction (nebulisers, epinephrine, steroids) (📖 p. 38)
- On discharge, ibuprofen 400–800 mg PO tid for one week, diphenhydramine 25 mg PO tid for one week. Topical treatment as above
- No IV steroids if no anaphylaxis

Critical documentation

- Location of insect bite, size and characteristics of skin/tissue reaction

Disposition

- Admit any patient with systemic symptoms
- Follow up 7–14 days. Be alert for secondary infection and serum sickness which can occur up to 14 days after initial sting
- If adrenaline autoinjectors are available, prescribe on discharge for any systemic reaction. If not, educate on prevention and recognition of anaphylaxis and prompt attendance to nearest health facility at earliest signs

89 Mammalian bites

Human, dog and cat bites are not uncommon and can lead to local and systemic infections if not addressed properly.

The first five minutes

- ABC, VS, O_2 as needed

History and physical examination

Key historical features

Date and time of injury.

- Animal bite:
  - What type of animal?
  - Does the patient know the animal/owner?
  - Is animal healthy?
  - Provoked vs. unprovoked
- Human bite:
  - Essential to elicit accurate history when hand wounds are caused by human bites as these have increased risk of deep tissue and bone infections

Signs and symptoms

- Establish blood-free area for examination, use tourniquet if needed
- Deeper wounds may need to be examined under anaesthesia ◇
- Location: inspect for puncture wounds and involvement of deeper structures such as neurovascular, tendon, or bone. For hand bites, inspect hand in both anatomical and position at time of injury
- Clenched fist against teeth (fight), usually causes small wound across dorsal surface of metacarpophalangeal joints
- Signs of local infection

Possible causes and differential diagnosis

- Other bites: insect bite, spider bite
- Cellulitis
- Laceration from fall or injury
- Consider child abuse and domestic violence

Investigations

This is a clinical diagnosis. Investigations are used to detect complications.

- XR ◇: to assess for foreign bodies, deeper tissue injuries, fractures
- US ◈: to assess for abscesses or effusion

Management

The goal of acute management is local wound care to prevent complications.

- Clean wound thoroughly. Use local anaesthetics
  - » Pressure irrigation
  - » Remove any foreign object
  - » Debride devitalised epidermal tissue
- Tetanus booster
- Rabies prophylaxis if indicated ◇ (📖 p. 349)
- Primary closure:
  - » Some dog wounds can be closed primarily:
    - ▪ Clean bite mark without signs of infection/less than 12 hours old
    - ▪ Not located on hand or feet
  - » Cat and human bite wounds are closed by delayed primary intention or secondary intention
  - » Bite wounds on face may be closed for cosmetic reasons once irrigated
- Do not suture: crush injuries, puncture wounds, bites on hands or feet, wounds older than 12 hours, cat or human bites (other than those on face) or wounds in immunocompromised patients:
  - » Antibiotic prophylaxis indicated for these injuries
- Animals bites: most common pathogens include *Pasteurella* species, staphylococci, streptococci, anaerobic bacteria and *Bartonella henselae*
- Human bites: polymicrobial from human saliva:
  - » Most common pathogens include *Eikenella corrodens, Staphylococcus, Streptococcus*, and *Corynebacterium* species
  - » Small risk of transmission of systemic disease: HIV, hepatitis, syphilis. Prophylactic treatment for HIV and hepatitis only in large wounds with bleeding inflicted by a known carrier
- Antibiotics should covers both aerobes and anaerobes: amoxicillin/clavuluate (Augmentin) or a second/third generation cephalosporin
- Consult surgery for wounds that need further exploration or debridement:
  - » Complex lacerations of head and neck
  - » Any involvement of tendon, bones, joints, neurovascular structures
  - » Infections

Critical documentation

Neurovascular examination: motor, sensory, tendon, pulse, range of movement.

Disposition

Admit patients with signs of systemic toxicity or infection ◇.

Most bite wounds can be assessed and discharged – teach patients to assess for signs of infection; follow up within 24–48 hours for wound check.

90 Scorpion stings

Many scorpion stings are harmless and cause only localised pain. However, highly toxic species are found. Most are nocturnal, prefer cool, dark places, and only sting humans when disturbed.

The first five minutes

- ABC, VS, resuscitate as needed
- Envenomation site should be immobilised and cold packs applied if available

History and physical examination

Key historical features

- Intense pain at the site of the sting – most older children and adults will give a history of a sting

Signs and symptoms

- Clinical symptoms due to release of neurotransmitters:
  - » Acetylcholine: vomiting, abdominal pain, bradycardia, diaphoresis, salivation
  - » Catecholamines: hypertension, tachycardia, pulmonary oedema, cardiac dysfunction
- In severe cases, cranial nerve and motor dysfunction can lead to respiratory compromise, agitation, uncontrollable extremity spastic movement, haemorrhage, skin necrosis, and death

Possible causes and differential diagnosis

Organophosphate poisoning, snakebite, spider bite.

Investigations

Scorpion stings are a clinical diagnosis, indicated by history and physical examination.

- Labs: electrolytes, CBC, renal function ◇ cardiac enzymes, lipase ◈
- ECG ◇: arrhythmia due to catecholamine release
- Imaging: CXR ◇ (pulmonary oedema); echo ◈ (dyskinesis, LV failure)

Management

The goal of acute management is prevention of systemic envenomation and pain control.

- Opiates or nerve block if available
- Immobilise the affected extremity
- Bandage that compresses lymphatic vessels and reduces systemic envenomation, immobilisation, and cold packs (if available) can slow absorption of the toxin
- Antivenin ◇ (where available, check package insert for dosing) – this should rapidly reverse cranial nerve dysfunction and muscular symptoms but has no effect on pain and paresthesias
  - » Watch for anaphylaxis. Treat with adrenaline (0.5 mg IM in adults or 10 µg/kg IM in children), IV diphenhydramine, and hydrocortisone 200 mg. Consider premedicating with these agents
- Hypertensive crisis/pulmonary oedema: vasodilators (nitroglycerine, hydralazine, nifedipine) ◇

- Bradyarrhythmias: atropine 0.5 mg IV
- Benzodiazepines should be administered for agitation and psychomotor symptoms
- Scorpion venom tends to spread rapidly in children and severe illness generally manifests quickly. Patients should be observed for signs of autonomic instability, myoclonus and pulmonary oedema for several hours

Critical documentation

Document initial and repeat VS, examination of the wound, pulmonary examination, and evidence of hypertensive crisis or pulmonary oedema

Disposition

Admit patients with systemic illness. Discharge asymptomatic patients once pain controlled.

91 Snake bites

Most snake bites in Africa occur in farm workers, women, and children in rural communities. Approximately 50% need treatment. Clinically relevant venomous snakes in Africa include:
- Elapidae (characterised by smooth scales, round pupils, short, fixed fangs – includes cobras, mambas with neurotoxic venom)
- Viperidae (long, hinged fangs, triangular-shaped head, many have elliptical shaped pupils, and many are nocturnal – includes pit vipers, adders with cytotoxic venom)
- Colubridae (rear fanged, 'boomslang' or tree snake with haemotoxic venom)

The first five minutes

Do not send family to retrieve snake. If brought in remember that biting (with envenomation) can occur after the snake has died.
- Reassure the patient – half of bites are 'dry' (no venom)
- Do not disturb the bite site (do not cut or suck venom from wound) or apply tourniquets
- Immobilise the person and wound as much as possible

History and physical examination

Key historical features

Description of snake and circumstances; consult local experts if available.

Signs and symptoms

- Proteolytic (cytotoxic) venom: (usually viper bites):
  - Local oedema, blistering, necrosis, evidence of compartment syndrome
  - Haemorrhage or ecchymosis
  - Myalgia, myoglobinuria (black urine) (evidence of rhabdomyolysis), decreased urine output (renal failure)
  - Hypotension or arrhythmias
- Neurotoxic venom (usually elapid bites – often no to minimal local symptoms and delayed presentation):
  - Descending paralysis that can impede bulbar and respiratory function
  - Tremors, salivation, dysarthria, diplopia, ptosis, fixed myosis, seizures
- Some cobras 'spit' venom at their victims, who may complain of eye pain, tearing, and impaired vision
- Haemotoxic venom: 'Boomslang' – slow acting haemotoxic venom that affects blood clotting and requires specific antivenom

Investigations

Measure limb circumference above and below the bite, mark border of oedema/erythema and reassess every 30 minutes. Check compartment pressures if compartment syndrome suspected.

• Labs: CBC, electrolytes, renal, urinalysis; clotting test (2 ml of venous blood, place in a clean tube and do not disturb for 20 min, then tip once. If blood flows out, coagulopathy has occurred.) ◇; PT/PTT ◈
• ECG ◇ (arrhythmias)
• Pulmonary function testing to evaluate for pulmonary involvement in neurological involvement ◈

Management

The goal of acute management is to prevent systemic envenomation.
• Immobilise the affected limb in a comfortable position.
• Wrap entire bitten limb tightly with compressive bandage to compress lymphatics. Loosen only for intolerable pain or signs of impeded circulation
• Give tetanus booster if needed ◇
• Give antibiotics if signs of infection or extensive tissue destruction
• Correct hypovolaemia, IV crystalloid for rhabdomyolysis or hypotension
• Debride necrotic tissue as necessary, but avoid fasciotomy unless clotting test has returned to normal
• Antivenin if any signs of rapid spreading, compartment syndrome, systemic toxicity
  » Formulation varies by local species, so check package insert for appropriate species covered and medication dosing. ◈ Watch for signs of anaphylaxis with antivenin administration (incidence as high as 14%). (See 📖 Anaphylaxis p. 38.)
• If signs of compartment syndrome, elevate the limb, administer mannitol 1–2 g/kg over 30 minutes while giving antivenin. Fasciotomy only if compartment pressures > 30 mmHg after antivenin given and coagulopathy improved.
• Respiratory paralysis or inability to control secretions/swallow requires intubation and ventilator support if feasible
• Dialysis may be necessary for acute kidney injury

Critical documentation

Document type of envenomation suspected; describe snake; estimate time of bite, antivenin given, any allergic reaction.

Disposition

Observe for at least 24 hours (signs of envenomation can take > 12 hours to present).

92 Spider bites

There are over 30 000 known different species of spiders globally, but only about 60 types are known to bite humans. Four spiders are dangerous to humans: 1) *Latrodectus* (widows) (e.g. black widow); 2) *Loxosceles* (violin or brown recluse); 3) *Harpactirinae* (African tarantula) (e.g. baboon spider); and 4) *Cheiracanthium* (sac spiders). Additionally, *Sicarius* (e.g. six-eyed crab, sand or assassin spiders) are highly venomous, but recorded bites are extremely rare.

Types

• Black widow spiders (button spiders):
  » Glossy black with red hourglass or stripe at the abdomen
  » Prefer dark environments such as garages and stables
• Brown recluse spiders (violin spiders):
  » Violin-shaped figure at mid-back with the neck of violin pointing to the rear
  » Prefer dark, warm environments such as closets and attics
• Tarantulas (baboon spiders):
  » Hairy, up to 150–250 mm long, colour range from rust to bright red
  » Aggressive, nocturnal burrowing spiders
  » Mildly neurotoxic venom

• Sac spiders:
  » Pale yellow spiders with black head
  » Cytotoxic venom
  » Cause of most cytotoxic spider bites in southern Africa

Signs and symptoms

• Black widow bites:
  » Localised pain, swelling and erythema
  » Muscle spasms, **abdominal cramping and rigidity**
  » Can progress to systemic toxicity from neurotoxic effects
  » Symptoms resolve within 2–3 days without complications
• Brown recluse bites:
  » Symptoms range from asymptomatic to systemic effects according to amount of venom injected
  » Localised pain, **bull's eye rash** with central bleb, **tissue necrosis**
  » Systemic effects including fever, chills, nausea, vomiting, malaise, weakness, **shock**, **haemolysis**, **DIC**, **renal failure** can occur
• Tarantula bites:
  » Intense burning, erythema and swelling at the bite site
  » Allergic and anaphylactic reactions to venom have been reported
  » Venom itself is not dangerous to humans
• Sac spiders:
  » Bite initially painless but progresses to painful, large lesion in several days
  » Lesion usually takes weeks to months to resolve

Diagnosis

Made by symptoms and identification of the spider when possible.

Investigations

Labs: CBC, electrolytes, renal, urinalysis ◇; PT/PTT ◈

Management

The goal of acute management is analgesia and management of systemic effects.
• Black widow spider:
  » Ice, analgesia, irrigate wound with soap and water
  » Tetanus vaccine
  » Benzodiazepines for abdominal cramping and rigidity
  » Antivenin for patients < 16 years or > 65 years, significant pain despite analgesics, unable to stand, with dangerous hypertension or pregnant women
• Brown recluse spiders:
  » Ice, analgesia, irrigate wound with soap and water
  » Tetanus vaccine
  » Haemodialysis for renal failure
  » Dapsone 50–200 mg/day may be beneficial
  » Antivenin if locally available for severe systemic toxicity
• Tarantula:
  » Symptomatic treatment
• Sac spider:
  » Oral antibiotics if secondary infection develops
  » Symptomatic treatment
  » No antivenin available

Disposition

Admit patients with severe toxicity, preferably to ICU. Admit elderly, pregnant, children and patients with serious comorbidities. Discharge well patients with mild symptoms, after six hours of observation.

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Part

4

F. Gastrointestinal

93 Approach to the adult with abdominal mass

Abdominal masses may represent enlargement of an abdominal or pelvic organ, neoplastic tissue (benign or malignant) or non-tissue collections (inflammatory masses, faeces, etc.). The location of the mass provides the first clue to diagnosis and is often the most important differentiating factor.

The first five minutes

- As needed – ABC, VS, O_2, monitors, glucose
- Rarely immediately life threatening (exceptions – ruptured AAA, infective masses in the context of septic shock)

History and physical examination

Key historical features

- Ask about the mass: when did the patient notice it, is it constant?

• Medical conditions, medication, pregnancy, family history of malignancy
• Associated features: pain, weight loss, fever, nausea/vomiting, change in bowel habits; GUT symptoms (dysuria, frequency, abnormal discharges or bleeding, amenorrhoea, haematuria) and other pertinent complaints (jaundice, flatulence etc.)

Signs and symptoms

• Overall features of malignancy (weight loss, anaemia, evidence of distant metastases)
• Detailed examination of abdomen, pelvis and back (including rectal and genital)
• Narrow differential by location (see Table 93.1)
• Differentiate abdominal wall vs. intra-abdominal masses. Have the patient contract the abdominal muscles: abdominal wall masses feel more prominent; intra-abdominal masses become less prominent
• Describe size, shape, consistency, tenderness, surface contour, percussion findings and dynamic features (peristalsis, pulsation)
• Tenderness often indicates infection/inflammation but is also associated with vascular compromise (leak, rupture, occlusion), tumour complications (tumour necrosis or infarction), local compression effects, gut stretching (faecal loading, obstruction)
• Tympany indicates a gas-filled mass (i.e. distended bowel); dullness indicates a solid or fluid filled mass
• Pulsatile masses are probably vascular while masses with peristalsis indicates bowel origin

Differential diagnosis

Table 93.1 The differential diagnosis of an abdominal mass in an adult

Right upper quadrant (RUQ): Hepatomegaly (hepatitis, abscess or tumour) Distended gallbladder (cholecystitis, pancreatic cancer, carcinoma)	**Epigastric:** Acute gastric distention Pancreatic pseudocyst Pancreatic cancer Abdominal aortic aneurysm	**Left upper quadrant (LUQ):** Splenomegaly Carcinoma of the splenic flexure
Right mid-abdomen: Renal cyst Polycystic kidneys Renal malignancies	**Periumbilical:** Bowel malignancy	**Left mid-abdomen:** Renal cyst Polycystic kidneys Renal malignancies
Right lower quadrant (RLQ): Appendicael abscess, caecal carcinoma Inflammation or neoplasm of the intestine	**Hypogastric:** Acute urinary retention Uterine or ovarian neoplasm (benign and malignant) Pregnancy	**Left lower quadrant (LLQ):** Diverticular abscess, sigmoid carcinoma, inflammation or neoplasm of the intestine
Any intra-abdominal location: Nematodes, trematoeds, cestodes,*Amoeba*, TB, lymphoma (especially Burkitt's)		
Abdominal wall masses: (Most common) hernias, neoplasm, infection, haematoma		

Investigations

Specific investigations will be guided by the suspected diagnosis. US is probably the most useful initial investigation. AXR is almost never helpful unless bowel obstruction suspected.
• Labs: CBC (infection), renal ◇; specific analyses may be helpful (e.g. LFT in RUQ masses)
• Imaging: AXR (bowel obstruction), US (fluid-filled masses with septae (ecchinococcus), abscess (amaeboma, bacterial abscess, cyst), worms or filariae, inflamed bowel wall (colitis, appendicitis), gallbladder pathology, etc.) ◇; contrast CT abdomen ◈

Management

The goal of acute management is symptomatic treatment and prompt diagnosis. Management is based on the specific origin of the mass.

Critical documentation

Detailed clinical and special investigation findings. Document any intervention, referral and follow-up offered to the

patient.

Disposition

Admit if danger signs: inability to tolerate per os, abnormal VS etc. Discharge other patients with follow-up.

94 Approach to the child with abdominal mass

Paediatric abdominal masses are a diagnostic challenge. Age is one of the most important factors in determining likely aetiology.

The first five minutes

• ABC, VS, IVF as needed
• Rarely immediately life threatening (exceptions include infective masses in the context of septic shock)

History and physical examination

Key historical features

Ask about:
• Time since mass was found, rapidity of growth
• Evidence of gastrointestinal or genitourinary obstruction
• Constitutional symptoms (pallor, anorexia, fever, weight loss, etc.) may suggest malignant lesion
• In neonates and young infants, information from prenatal history can be helpful. The presence of oligohydramnios or polyhydramnios on prenatal US might suggest disease affecting the developing fetal renal system

Signs and symptoms

A thorough examination can be difficult in the very young or uncooperative child.
• Auscultate for bowel sounds (intestinal obstruction)
• Percussion helps detect organ or mass size and assists in differentiating air filled from solid
• Assess for guarding or tenderness (inflammatory or infectious process)

Differential diagnosis

Varies by age. Neonates: over half of palpable masses in neonates originate from the genitourinary tract. Hydronephrosis and multidysplastic kidney are the most common. Malignant lesions are more common in infants and children than in neonates. The most common tumours are neuroblastomas, Wilms' tumours, and lymphomas. GI parasites and abscesses can also present as abdominal masses (📖 p. 354).

Table 94.1 Non-infectious causes of abdominal mass in neonates, infants and children

Neonates	Infants and children
Renal/Retroperitoneal	
Hydronephrosis Multidysplastic kidney Mesoblastic nephroma Renal vein thrombosis Polycystic kidney disease Wilms' tumor Rhabdoid tumor Adrenal masses	Neuroblastoma Wilms' tumor Lymphoma Liver Haemangioma or cyst Hepatoblastoma Embryonal sarcoma Polycystic kidney disease
Pelvic	
Ovarian cyst Hydrocolpos Hydrometrocolpos	Ovarian cysts Teratomas

Gastrointestinal	
GI duplication Gastroschisis Omphalocoele	Duplication Bowel obstruction Meckel's diverticulum Faecal mass

Investigations

Laboratory investigations should be directed to the specific cause.
• Imaging: AXR (bowel obstruction, calcified mass), US (often diagnostic) ◇; CT (definitive diagnosis)

Management

The goal of acute management is symptomatic treatment and prompt identification of the cause. Management is then based on the specific origin of the mass. Involve surgery, urology, oncology early.

Critical documentation

Detailed clinical and special investigation findings. Document any intervention, referral and follow-up offered to the patient.

Disposition

Admit if danger signs, evidence of obstruction, or inability to tolerate per os. Discharge other patients with follow-up.

95 Appendicitis

Appendicitis is inflammation and/or infection of the appendix. It is the most common surgical cause of abdominal pain in children and adults < 50 years. It is rare but more severe in children < 5 years. Incidence of appendicitis is unchanged by pregnancy, but the diagnosis is more difficult and complications such as miscarriage more likely.

The first five minutes

- ABC, VS, O_2, IV, monitors, glucose. Evaluate for signs of sepsis
- Keep NPO, fluid resuscitate as needed, provide analgesics and anti-emetics

History and physical examination

Key historical features

No single sign, symptom or test accurately confirms appendicitis in all cases. Most patients present with some combination of abdominal pain, fever and nausea. The 'classic' presentation is cramp-like peri-umbilical pain that migrates to the right lower quadrant. Common associated features include nausea, vomiting, and fever. Ask about genitourinary symptoms. A detailed gynaecological history is always required in women. Children often present earlier in their clinical course, when only mild or less specific symptoms are present. Elderly and chronically ill patients may present late with a more non-specific and indolent course.

Signs and symptoms

- Detailed examination of the abdomen, pelvis and back, rectal and vaginal/genital; check males for testicular torsion
- Abdominal examination may reveal tenderness over McBurney's point
- Look for evidence of peritonism (guarding, percussion tenderness, rebound)

Differential diagnosis

Includes any cause of abdominal pain and tenderness. Pelvic inflammatory disease, ectopic pregnancy, gastroenteritis and bowel perforation; consider pancreatitis, cholecystitis, mesenteric adenitis, mesenteric ischaemia, omental torsion, biliary colic, renal colic, urinary tract infection (UTI), diverticulitis, Crohn's disease, CGIT malignancy, endometriosis, ovarian cyst or torsion, testicular torsion.

Investigations

Laboratory tests are not specific for appendicitis.

- Labs: CBC (leucoytosis with left shift), urinalysis, pregnancy test, LFT ◇; lipase/amylase ◈
- Imaging: US abdomen (sensitivity 85% specificity > 90% in experienced hands) ◇; contrast CT abdomen (more accurate) ◈. A classic presentation in a younger person (especially male) does not require CT confirmation, and unnecessary radiation should be avoided

Management

The goal of acute management is prompt diagnosis, fluid replacement, analgesia, and early surgical consultation. If the diagnosis is clinically obvious, surgical referral is mandatory.

Management is classically appendectomy; recent evidence suggests that mild cases may be treated with PO antibiotics alone. Symptomatic treatment (analgesia, anti-emetics) is essential.

Critical documentation

Age, key history and serial examination findings, investigations, management and surgical consultation communication.

Disposition

Most are admitted.

Table 95.1 Clinical features in appendicitis

Feature	Sensitivity	Specificity
Symptoms		
RLQ pain	0.81	0.53
Migration	0.64	0.82
Pain before vomiting	1.00	0.64
Anorexia	0.68	0.36
Vomiting	0.51	0.45
No similar pain previously	0.51	0.41
Signs		
Fever	0.67	0.79
Rebound tenderness	0.63	0.69
Psoas sign	0.16	0.95
Rigidity	0.27	0.83
Rectal tenderness	0.41	0.77

Source: Wyatt et al, *Handbook of Emergency Medicine*, Oxford University Press USA

96 Bowel obstruction

Bowel obstruction is a mechanical or functional blockage of the gastrointestinal tract which causes decreased transit of bowel content. There are many types and levels of obstruction but all are serious and associated with poor outcome if not managed properly.

The first five minutes

- ABC, O_2, IV access, monitor, glucose

• Evaluate for sepsis or shock

History and physical examination

Key historical features

Features of decreased output below the obstruction (constipation, obstipation), features of obstruction to inflow above (vomiting, abdominal distension, pain), increased peristalsis (classic crescendo-decrescendo cramp-like pain), features of complications (shock, sepsis, perforation, electrolyte abnormalities, bowel necrosis) and possibly features of the cause (evidence of previous bowel surgery or trauma, malignancy etc.).

Signs and symptoms

• Look for features of sepsis and perforation (fever, tachycardia, peritonism)
• Overall appearance for dehydration or haemodynamic compromise
• Detailed examination of the abdomen
• Classic findings include a tympanically distended abdomen, initial hyperperistalsis (increased bowel sounds and visible bowel movements) and eventual hypoperistalsis (decreased bowel sounds)
• Evaluation for a possible hernia or abdominal mass
• Digital rectal exam may reveal an empty rectum, a mass or impacted faeces

Differential diagnosis

• Common causes of large bowel obstruction: malignancy and faecal impaction
• Common causes of small bowel obstruction: adhesions and malrotation
• Also consider: herniation, inflammatory bowel disease, intussusception, volvulus, stricture, foreign bodies, ascaris, etc.
• Functional obstruction (hypo-motility) is usually caused by electrolyte abnormalities (hypokalaemia), severe systemic illness, over-use of laxatives or peritonitis
• Perforated peptic ulcer disease with illeus.

Investigations

• Labs: electrolytes, renal ◇
• Imaging: AXR (accuracy 60–80%; classic findings – dilated bowel, air-fluid levels, bowel markings and no air in the rectum (or paucity of distal gas); small bowel obstruction – air-fluid levels and dilated loops of bowel centrally located and horizontally orientated; small bowel markings are fine and cross the entire width of the lumen (valvulae conniventes); large bowel obstruction – air-fluid levels and dilated bowel is usually peripheral and vertical; colonic haustral lines do not cross the entire width of the lumen; sigmoid volvulus – a dilated loop of large bowel extending from the pelvis while cecal volvulus extends from the right lower quadrant), erect CXR (evidence of bowel perforation (free air under the diaphragm)); US (dilated bowel, sensitivity 85% in experienced hands) ◇; CT (90% sensitive for high-grade obstructions, can determine the level, severity, and perhaps cause of obstruction) ◈

Management

The goal of acute management is fluid replacement, analgesia and early NGT for decompression. Consult surgery for operative decompression or conservative management with NGT and IVF. Risk of perforation increases with increased lumen pressures, so time to decompression is critical.

Critical documentation

Document clinical and special investigation findings, initial management, time of referral and evaluation by the surgical team and the disposition decision.

Disposition

Admit all patients to surgery.

97 Anal and rectal disorders

Anal and rectal disorders include a diverse number of diagnoses including functional, structural and infectious aetiologies.

The first five minutes

ABC, VS, check for haemorrhage.

History and physical examination

Key historical features

PR bleeding, change in bowel habit, GU symptoms.

Signs and symptoms

Exam must include abdominal exam, anal and perinial inspection, digital rectal examination and anoscopy ◇.

Differential diagnosis

Functional

Causes of faecal impaction include constipation, pelvic pain with episodic spasm, passage of liquid stool only (passed around the obstructing faecal bolus), abdominal distension.

Structural

- Fissure – result of excessive stretching of anal canal. Tearing pain on defecation, may have bright red blood
- Haemorrhoids:
  - Internal – rectal fullness and discomfort, bright blood, mucus
  - External – perianal mass, sudden, severe pain; purple, oedematous, subcutaneous perianal mass on exam
- Fistula – can arise from obstetric injuries, Crohn’s disease, complication of infection, trauma, radiation, tuberculosis and malignancy:
  - Rectovaginal fistula – passage of stool and flatus from the vagina
  - Fistula-in-ano – chronic mucopurulent discharge after abscess
- Neoplasm – chronic irregular edged, perianal ulceration, mass, bleeding, pain, discharge, tenesmus
- Rectal prolapse – incontinence, mucus discharge, pain, bleeding with mass of prolapsed tissue on exam

Infectious

- Proctitis/anusitis – rectal bleeding, discharge, rectal pain or painless ulceration/growth
- Abscess – anal (perirectal) or sacrococcygeal pain (pilonidal), tenderness, purulent discharge; tender fluctuant mass on exam
- Condylomata – fungating mass, often associated with immunocompromise.

Investigations

Most diagnoses are based on clinical exam and history.

Biopsy and staging with CT is required for neoplasms ⟐.

Management

Faecal impaction

Disimpaction and aggressive bowel regimen with stool softeners and enemas.

Fissure

Most respond to conservative management – pain relief, constipation control, warm baths. Topical nitroglycerin or PO nifedipine ◈ may help.

Haemorrhoids

Dietary alternations with bulking agents, stool softeners, increased fluids. Surgical excision for severe pain, necrosis, bleeding ◈.

Fistula

Recto-vaginal fistula and fistula-in ano requires surgical fixation after treatment of any underlying disease (Crohn's, abscess etc.) ◈.

Rectal prolapse

Reduction of the prolapsed portion with use of topical sugar application to reduce bowel oedema. Surgical repair (rectopexy) is indicated for recurrent disease or refractory prolapse.

Proctitis/anusitis

Identify the most likely cause based on exam (syphilis, gonococcal, LGV, herpes, choncroid) and treat according to local treatment guidelines.

Abscess

Pilonidal: I+D under local anaesthesia; perirectal abscess require operative drainage.

Critical documentation

Document detailed clinical findings, VS, response to therapy.

Disposition

Discharge and follow-up uncomplicated anorectal disorders. Admit to surgery if abscess or requires surgical management.

98 Approach to the patient with ascites

Ascites is an abnormal collection of fluid in the peritoneal cavity. This fluid accumulates due to a variety of pathological mechanisms. Ascites may indicate a dangerous cause (malignancy, organ failure) or pose danger itself (secondary infection, large volume with respiratory compromise).

The first five minutes

ABC, VS, O_2, glucose if AMS.

History and physical examination

Key historical features

- Ask about fever, constitutional symptoms and weight loss
- Patients may report increased abdominal girth, weight gain, lower extremity oedema, abdominal pain, nausea, vomiting, diarrhoea or shortness of breath

- Ask about liver disease or potential causes of liver failure: portal hypertension, alcohol abuse, ascites, NSAID/steroid use, malignancy, viral hepatitis or TB
- Any AMS (hepatic encephalopathy)

Signs and symptoms

Assess for masses, skin changes (caput medusa, spider angiomata). Palpate for tenderness, signs of peritonitis, hepatomegaly or splenomegaly. Evaluate for bulging flanks, flank dullness, shifting dullness, fluid wave. Evaluate for signs of hepatic encephalopathy.

Differential diagnosis

- Transudate from the splanchnic circulation due to increased portal venous pressure (commonest mechanism): cirrhosis, hepatitis, hepatic veno-occlusive disease, liver metastasis, CHF, constrictive pericarditis
- Low serum oncotic pressure: nephrotic syndrome, malnutrition, mal-absorption
- Other causes: obstruction of normal lymphatic drainage, increased production of peritoneal fluid due to inflammation, TB, peritoneal carcinomatosis

Investigations

- Labs: CBC, electrolytes, renal, LFTs ◇; PT/PTT ◈
- Ascitic fluid analysis is usually diagnostic, includes cell count and differential, gram stain and culture, ≥ 250 PMN/mm3 is diagnostic for spontaneous bacterial peritonitis ◇
- Imaging usually not diagnostic: AXR (rule out bowel obstruction), US (can helpful to identify peritoneal fluid and guide paracentesis if exam is difficult) ◇

Management

The goal of acute management is symptomatic relief and prevention of infection.

Paracentesis

(See 🕮 p. 848.)

Pharmacotherapy

May respond to dietary sodium restriction and diuretic therapy with aldosterone antagonist such as spironolactone ◇ with or without addition of furosemide ◇.

Spontaneous bacterial peritonitis

- High mortality in setting of cirrhosis
- Treatment: ceftriaxone (or similar) five days. The use of albumin is not supported by recent evidence
- Prevention: long term prophylaxis with quinolone

Definitive management

Patients refractory to low sodium diet and high-dose diuretics can be considered for serial therapeutic paracentesis, TIPS procedure ◈ or liver transplant ◈.

Critical documentation

Document detailed history and physical, vital signs, procedures, frequent reassessments, response to therapy, expert consultation, and laboratory results.

Disposition

Admit patients with danger signs, evidence of new liver failure and sepsis, and all cases of SBP.

99 Approach to diarrhoea

Diarrhoea is defined as increased frequency of stool (> 3 per day), often accompanied by increased volume and change in consistency. Most cases are caused by infection or toxins; other causes include non-infective inflammation (IBS), malabsorption, hyper-motility, drug side effects. In children, especially those suffering from micronutrient deficiency, diarrhoea can be severe and fatal.

Acute diarrhoea has a sudden onset and lasts ≤ 14 days; chronic/persistent diarrhoea lasts > 14 days. Most cases are harmless and self-limiting but dangerous complications such as dehydration, shock, bowel perforation and sepsis can occur.

The first five minutes

- ABC, VS, IVF, glucose
- Identify severe dehydration or shock

History and physical examination

Key historical features

- Diarrhoea: onset, duration, volume, frequency and abnormal stool content (blood, mucus or pus may indicate a dangerous infective cause)
- Associated features: fever, vomiting, pain, jaundice and weight loss
- Co-morbid illness (particularly HIV), medication, and allergies
- Possible causes: travel history, dietary change, and sick contacts with similar symptoms or disease outbreaks (ask specifically about cholera)

Signs and symptoms

- Evaluate for dehydration, malnutrition. In children evaluate skin turgor, sunken eyes, level of consciousness
- Fever, haemodynamic status, hydration
- If bloody stool, do a rectal exam

Differential diagnosis

Acute diarrhoea

- Fever, blood: acute bacillary dysentery, *Campylobacter* enterocolitis, *Salmonella* enterocolitis, *Shigella* dysentery, EHEC1
- Fever, no blood: *Salmonella* enteritis, mild *Shigella* enteritis, *Campylobacter* enteritis, ETEC1
- No fever, blood: amoebic dysentery, *S. mansoni*, ulcerative colitis, *C. difficile* enteritis
- No fever, no blood: cholera, food poisoning, toxins/poisons, staphylococcal/*Clostrididrium*

Chronic diarrhoea

Consider HIV/AIDS, inflammatory bowel disease, medication side effects (prolonged antibiotic use), diabetes, food sensitivities, malabsorption and malignancy.

Investigations

Most do not require investigations, which when done should be directed towards complications (e.g. severe dehydration, shock or confusion: electrolytes, renal, consider evaluation for sepsis) and finding the cause (e.g. features of dysentery: stool and possibly blood cultures; chronic, treatment resistant diarrhoea (especially if HIV positive) – stool for (parasites, protozoa, bacteria).

Management

The goal of acute management is fluid resuscitation, commencement of specific therapies (where indicated) and determination of cause.

- Oral rehydration is the cornerstone of managing dehydration. In children who are breastfeeding, encourage mother to continue
- Administer IVF if severely dehydrated and unable to tolerate PO. For adults: 1–2 l bolus. For children: see RAP Dehydration p. 50 and Volume resuscitation in children p. 27
- Antibiotics targeted to the suspected organism
- Avoid anti-motility agents in patients with dysentery or other evidence of invasive bacterial/protozoan disease
- In children consider zinc supplementation for 10–14 days: 10 mg/day if < 6 months, 20 mg/day if > 6 months
- Isolation must be considered if a highly infectious cause (i.e. cholera) is suspected. Notify health authorities per local regulations

Critical documentation

Key history and exam findings emphasising hydration status and signs that point towards a specific aetiology; treatment plan indicating fluid replacement type, route and review periods; investigations ordered and results if available; follow-up plan if managed as outpatient.

Disposition

Admit for severe dehydration or shock, suspected sepsis, severe hypokalaemia, unable to tolerate PO and haemodynamic instability. Discharge cases and educate on oral rehydration and infection prevention procedures. Return if persists > 10 days, bloody stools, fever, worsening abdominal pain, severe vomiting or worsening dehydration.

100 Gallbladder disease

The most common conditions that affect the gallbladder and bile ducts are biliary colic (cholelithiasis – pain related to mechanical effects of gallstones), cholecystitis (inflammation or infection), cholangitis (infection of the bile ducts) and choledocholithiasis (obstructive stone in the common bile duct). Also consider malignancy, parasitic infestation and auto-immune inflammation with fibrosis. For ascending cholangitis, see Jaundice in adults, p. 264.

The cardinal features of gallbladder and bile duct disease are:

- Pain: usually severe; colicky with gallstone disease, constant with infection or total stone impaction
- Jaundice: obstructive pattern if the CBD is obstructed
- Anorexia and malaise
- Fever (with infection)

Common and dangerous complications include: systemic sepsis, perforation with peritonitis and abdominal abscesses/sepsis, pancreatitis.

The first five minutes

- ABC, VS, O_2, IV, glucose
- Identify and treat those with sepsis/septic shock

History and physical examination

Key historical features

- Pain in right upper quadrant/epigastrium, with radiation to back, right shoulder/clavicular area
- Biliary colic presents with severe, colicky, intermittent pain
- Cholecystitis: classically characterised by persistent RUQ pain, nausea/vomiting, fever; previous episodes of biliary colic in many patients

• Important comorbid conditions, previous GB disease or surgery

Signs and symptoms

• Look for fever, tachycardia, hypotension, jaundice and confusion
• RUQ tenderness and peritonism suggests cholecystitis. Generalised peritonism suggests perforation. Consider pancreatitis if upper abdominal pain is severe, constant and not accompanied by significant tenderness
• Murphy's sign (cholecystitis) – pain caused by deep breathing with the examiner's hand in the RUQ, fingers directed under the costal margin
• Look for features that may suggest malignancy or HIV infection (weight loss, anaemia etc.)

Differential diagnosis

Peptic ulcer disease, pancreatitis, aortic pathology, pyelonephritis/nephrolithiasis, liver pathology, acute coronary syndromes, basal pneumonia.

Investigations

• All patients should have glucose tested and abdominal US (if available). Further tests depend on the clinical scenario
• Labs: WBC, LFT of limited utility. Evaluate for sepsis, pancreatitis and liver dysfunction
• Imaging: AXR (rarely diagnostic; evaluate for bowel obstruction), US □ (gallstones, gallbladder wall thickening > 4 mm, enlargement (> 8 cm long-axis, > 4 cm short axis), pericholecystic fluid, sonographic Murphy's sign (tenderness with direct compression of gallbladder fundus by probe) suggestive)

Management

The goal of acute management is analgesia, stabilisation and identification and treatment of complications (pancreatitis, obstructive jaundice, GB perforation). Further treatment depends on the cause.

Cholecystitis

Initial stabilisation with IVF, supportive care.
• Antibiotics: dictated by local sensitivities:
  » Cover gram negatives (*E. coli, K. pneumoniae*) and anaerobes (*B. fragilis*)
  » In mild to moderate disease, any cephalosporin, amoxicillin-clavulanate or ciprofloxacin ± metronidazole
  » ± Fluconazole if concern for *Candida* infection

Definitive management is surgical removal, usually after a period of IV antibiotics. Where available, biliary drainage is an alternative in the critically ill.

Biliary colic

Adequate analgesia is essential. If no complication and the pain resolves, outpatient follow-up and management is appropriate. Refer all other patients to surgical service.

Critical documentation

Clinical findings, especially presence or absence of Murphy's sign (most helpful physical exam finding) and fever.

Disposition

Discharge if pain controlled and tolerating fluid per os. Admit unstable patients to surgery.

101 Upper gastrointestinal bleeding

Bleeding proximal to the ligament of Treitz (includes oesophagus, stomach and duodenum) may be brisk, causing

shock, or chronic, causing anaemia. It may indicate underlying malignancy, especially in patients > 55 years, and those who smoke, drink alcohol or with untreated *H. pylori* infection. Consider also non-GI source (epistaxis with ingestion). See 🕮 RAP GI bleeding p. 66.

The first five minutes

- ABC, VS, 2 large bore IV, O_2, cardiac monitor, glucose
- For brisk bleed or abnormal VS, 2 l NS bolus followed by blood products

History and physical examination

Patients present with haematemesis, coffee-ground emesis, melaena. Severe bleeding may also present with shock and haematochezia.

Key historical features

- Bleeding: quantity, duration, prior episode(s), medications
- Associated symptoms: dizziness, weakness, syncope, pain, dysphagia, chest pain, dyspnoea
- Colour and quality: bright red or dark red/black (coffee-ground)
- Ask about medical history: liver disease, portal hypertension, alcohol abuse, PUD, chronic NSAID or steroid use, previous surgery, malignancy, hepatic schistosomiasis, aortic aneurysm. Cirrhosis and portal hypertension should raise concern for oesophageal variceal bleed which can decompensate rapidly
- Did forceful non-bloody emesis precede bleeding? Consider Mallory-Weiss tear

Signs and symptoms

- Evaluate for signs of shock and acute anaemia
- Abdominal tenderness: rebound or guarding, stigmata of portal hypertension, liver disease
- Rectal and stool exam for melaena

Differential diagnosis

PUD is the most common, other causes include upper GI malignancy, oesophageal or gastric varices, esophagitis, Mallory-Weiss tear, Boerhaave Syndrome, arteriovenous malformation, aortoenteric fistula.

Investigations

- Upper endoscopy if available ◈
- Labs: CBC, electrolytes, renal, LFT, type and cross ◇; PT/PTT ◈
- ECG ◇
- Imaging: upright CXR (evaluate for aspiration and perforation) ◇

Management

The goal of acute management is restoration of perfusion, identification of source and treatment of complications.
- Consider intubation for airway control ◇
- IVF and/or emergency transfusion for evidence of haemodynamic compromise or severe anaemia
- Tamponade with a Foley catheter or Sengstaken-Blakemore tube ◈
- Seek urgent expert consultation (for endoscopy or surgical management) in all patients with ongoing bleeding, haemodynamic compromise or severe anaemia ◈

Pharmacotherapy

- PPI 80 mg IV bolus, then 8 mg/hr infusion for 72 hours ◈
- If variceal bleeding, give terlipressin or octreotide ◈ and prophylactic quinolone or ceftriaxone.

Transfusion guidelines

Patients with massive bleeding should be transfused with blood, platelets and plasma according to local protocols.

- Transfuse for Hgb < 6 g/dl or < 8 g/dl with signs or history of ischaemia, or ongoing bleeding
- Transfuse platelets for active bleeding, if platelets < 50 x10^9/L
- Transfuse fresh frozen plasma (FFP) INR > 1.5 times normal

Definitive management

- Unstable patients may require emergency surgical resection if endoscopy is unavailable

Critical documentation

Serial and orthostatic VS, response to therapy, consultation, and serial Hgb.

Disposition

Admit for abnormal VS, anaemia, high BUN, coagulopathy, shock, ongoing bleeding, cirrhosis, or significant comorbidity.

102 Lower gastrointestinal bleeding

Lower GI bleeding (GIB) arises distal to the ligament of Treitz. It may cause anaemia, hypotension or shock, and may reflect underlying GI malignancy. Note that brisk upper GIB is a very common cause of lower GIB. See RAP GI bleeding p. 66.

> Massive lower GI bleeding is defined as:
>
> - Passage of a large volume of red or maroon blood from the rectum
> - Haemodynamic instability or shock
> - Significant drop in Hb (to 6 g/dl or less)
> - Bleeding requiring transfusion, continuing > 3 d, or re-bleeding within 1 week

The first five minutes

ABC, VS, O_2, IV access, monitors. Rectal exam to evaluate for massive bleeding.

History and physical examination

Key historical features

- Onset, duration, and quantity. Haematochezia (undigested blood mixed with stool) or melaena (partly digested blood, usually black, tarry and foul smelling)
- Prior episodes or any other bleeding
- Weight loss, change in bowel habits, abnormal bleeding, trauma
- Ask about associated symptoms (pain, dizziness, nausea etc.)
- Prior medical conditions, medication (aspirin or other blood thinners), allergies and a family history of cancer, bleeding disorders or vascular abnormalities

Signs and symptoms

- Features that indicate severity (e.g. anaemia, hypotension, exertional dyspnoea)
- Features of malignancy (weight loss, anaemia, evidence of metastases)
- Search for organomegaly, masses, tenderness and evidence of previous surgery or trauma
- Rectal exam: confirm bleeding. Look carefully for haemorrhoids, fissures and other local lesions

Differential diagnosis and possible causes

Diverticular disease is the most common (increases with age), other causes include infections or inflammatory bowel disease, angiodysplasia, colonic polyps, GIT malignancy, haemorrhoids, anal fissures, brisk upper GI bleed.

Investigations

Testing is guided by the clinical picture.

- Labs: Hgb, type and cross ◇
- Imaging: abdominal US, CXR (may reveal evidence of malignancy or perforation), GI contrast studies ◇; CT abdomen (may identify aetiology) ◈
- Anoscopy, sigmoidoscopy, colonoscopy ◈

Management

The goal of acute management is to manage shock and blood loss with fluid resuscitation, blood transfusion and urgent surgery as needed.

Transfusion guidelines

Patients with massive bleeding should be transfused with blood, platelets and plasma according to local protocols.

- Consider transfusion for Hgb < 6 g/dl or < 8 g/dl with incidence or history of cardiac disease, or ongoing bleeding
- Transfuse platelets for active bleeding, if platelets < 50 x 10^9/litre ◈
- Transfuse for FFP INR > 1.5 times normal ◈

Definitive management

Diverticular bleeding will stop spontaneously in over 80% of patients but will recur in approximately 25%. Unstable patients may require emergency surgical resection ◇. Consider interventional radiology if available ◈.

Critical documentation

Serial and orthostatic VS, response to therapy, consultation, and serial Hgb.

Disposition

Admit patients with massive lower GIB, high urea/creatinine, coagulopathy or significant comorbidity.

103 GI bleeding in children

Causes of GIB in children range from benign mechanical causes (constipation) to serious underlying pathology causing bleeding that may rapidly progress to shock. Always consider non-GI sources (epistaxis or haemoptosis with ingestion), or ingestion of blood from mother’s breast in breast-feeding infants.

The first five minutes

- ABC, VS, O_2, IV, monitor, glucose
- Start IVF early (20 ml/kg crystalloid in shocked child) followed by blood

History and physical examination

Key historical features

- Timing and duration, colour and quantity, prior symptoms, history of straining, abdominal pain, trauma, recent cough or epistaxis, any other bleeding (cogulopathy)
- Ask about food (e.g. chocolates, red liquorice,) or use of drugs (e.g. iron supplements, certain antibiotics, etc.)

that may give stool a 'bloody' appearance
• Ask about dry or cracked nipples in breast-feeding mothers

Features that may help to localise the site:
• Haematemesis = upper GI bleed
• Bright red blood coating stool = anorectal
• Haematochezia (fresh undigested blood mixed within stool) = the distal small bowel or proximal colon, occasionally severe acute upper GI
• 'Currant jelly' stools = vascular congestion and hyperaemia (often seen with intussusception)
• Frankly bloody diarrhoea usually reflects colonic bleeding
• Bright red blood mixed with mucus = colitis (infective or inflammatory)
• Melaena (digested blood mixed with stool) = upper GI bleed to ileo-cecal valve, above the duodenojejunal junction

Signs and symptoms

• Features that indicate severity (e.g. anaemia, hypotension)
• Features of malignancy (weight loss, anaemia, evidence of distant metastases, etc.)
• Organomegaly, masses, tenderness and evidence of previous surgery or trauma
• Rectal exam: search for tumours and confirm the bleeding. Also look carefully for, fissures, other local lesions and empty rectum

Differential diagnosis

Table 103.1 Differential diagnosis of GI bleed in children

Age	Upper GI	Lower GI
Neonates	Swallowed maternal blood, haemorrhagic disease of the newborn, coagulopathy, oesophagitis, gastritis, gastroduodenal ulcers and duplication cysts.	Swallowed maternal blood, anorectal fissures, necrotising enterocolitis, malrotation with midgut volvulus, coagulopathy and Hirschsprung's disease. Introduction of food products like milk or soy can cause enterocolitis or allergic colitis. Maternal use of medications like aspirin, cephalothin, and phenobarbital can cause coagulopathies.
1 month–1 year	Swallowed maternal blood, haemorrhagic disease of the newborn, coagulopathy, oesophagitis, gastritis, gastroduodenal ulcers and duplication cysts.	Intussusception presenting with episodic cramping abdominal pain, vomiting and currant jelly stools. Milk protein allergy may present with GI bleed, fussiness and increased bowel movements.
1–2 years	Peptic ulcers induced by NSAID use, and systemic diseases like burns (Curling ulcer), head trauma (Cushing ulcer), malignancy, or sepsis.	Anorectal fissures, allergic colitis (cow's milk protein allergy), intussusception, Merckel's diverticulum, gastrointestinal duplication, polyps, and ischemic bowel secondary to volvulus.
> 2 years	Mallory Weiss tears, oesophageal varices, gastric varices, gastritis, peptic ulcer disease	Infectious diarrhoea, juvenile polyps, inflammatory bowel disease, vascular lesions, HUS-HSP.

Investigations

• Labs: serial Hgb (much greater utility than single value), stool evaluation (leukocytes, bacterial culture, ova and parasites) ◇; *Clostridium difficile* toxin ◈
• Imaging: AXR (perforation or obstruction)
• Endoscopy ◈

Management

The goal of acute management is to stop bleeding and stabilise haemodynamics. Treatment directed at the underlying cause. Involve paediatric and surgical teams early.

Critical documentation

Age, estimation of blood lost and characteristics of bleed, serial VS and Hgb, response to therapy, consultation.

Disposition

Admit most patients, including all with abnormal VS, severe anaemia, hypovolemia, or when bowel obstruction is suspected.

104 Hernias

An abdominal hernia is a protrusion of any organ through a defect in the abdominal wall. The hernia sac and content may present as intermittent mass, or may become trapped leading to pain, obstruction, ischaemia and ultimately necrosis and perforation. 'Incarcerated' hernias cannot be reduced to proper location and may become strangulated (ischaemic).

Types of hernia

- Inguinal:
  - Indirect: most common groin hernia in both sexes, results from persistent process vaginalis. Hernial sac descends to scrotum in males
  - Direct: weakness in the transversalis fascia
- Femoral: more common in women:
  - Located inferior to the inguinal ligament through femoral ring
  - More likely to present as an incarceration or strangulation
- Umbilical: children (anatomical weakness at umbilical ring) and adults (due to increased abdominal girth, herniates through umbilical canal)
- Incisional: hernia at site of prior abdominal surgery
- Epigastric: located along the abdominal midline between the xiphoid and umbilicus due to weakness in linea alba

The first five minutes

- ABC, VS, IV access. Evaluate for incarceration
- Evaluate and treat for sepsis or shock, provide analgesia

History and physical examination

Key historical features

- Pain, mass, aggravating or alleviating factors
- Bowel obstruction: severe pain, vomiting, distension, constipation
- Evidence of perforation (fever, tachycardia, peritonitis, sepsis)
- Risk factors: personal or family history of hernia, history of abdominal surgery or trauma or AAA, male sex, smoking, obesity, pregnancy, heavy lifting, or chronic cough/constipation
- Specific risk factors in children: prematurity and low birth weight, increased intra-abdominal pressure (VP shunt, ascites, peritoneal dialysis)

Signs and symptoms

Hernias present as protrusions on the abdominal wall that may be reducible or not. May be easier to elicit with valsalva cough.

- Scrotal exam: swelling, pain, discoloration, audible bowel sounds
- Incarcerated (unable to reduce; vascular supply not compromised); painful swelling or fullness at hernia site, cannot be reduced with gentle pressure, symptoms/signs of bowel obstruction may be present
- Strangulated (unable to reduce with vascular supply compromise, eventual necrosis and perforation); toxic appearance, abdominal distension, bilious vomiting, discoloration of the entrapped viscera, pain out of proportion to exam

There are special considerations depending on the location in children (see Table 104.1).

Table 104.1 Special considerations in children

Type	Abdominal wall defect	Special considerations
Inguinal, indirect	Persistent process vaginalis	Palpate both testicles to rule out undescended or retractile.
Inguinal, direct	Fascial weakness of Hesselbach's triangle	
Femoral	Via the femoral ring	
Umbilical	Weakness at the umbilical ring	Low risk of incarceration, likely to self-resolve
Omphalocoele	Periumbilical; intestines covered by peritoneum	Emergency surgical consult required. Mortality depends on defect size, organ involvement, chromosomal abnormalities
Gastroschisis	Periumbilical; intestines exposed	Emergency surgical consult required. Mortality depends on intestinal dysfunction and obtaining wound closure
Diaphragmatic	Diaphragm	Abdominal contents herniate into the chest causing cardiac shift. Emergency surgical consult required. Mortality 40–62%

Differential diagnosis

Testicular pathology (torsion, infection, mass, hydrocele); any other cause of peritonitis or bowel obstruction.

Investigations

Clinical diagnosis. Lab tests are of little utility, but may reveal associated electrolyte abnormalities or renal dysfunction.

- Imaging: AXR (obstruction or bowel air outside the abdominal cavity); US (differentiate groin or abdominal wall masses) ◇; CT (confirm diagnosis and assess incarceration or strangulation) ◈

Management

The goal of acute management is to stabilise the patient, and identify and manage complications. Attempt reduction with patient in Trendelenburg, applying sustained gentle pressure.

- Reducible or asymptomatic: advise on lifestyle changes (such as avoiding heavy lifting and strenuous activities/exercise). Outpatient surgical referral.
- Incarcerated: attempt to reduce with analgesia and sedation
- Strangulated (or toxic appearing): do not manually reduce the hernia (avoid introducing necrotic bowel into abdominal cavity). Emergency surgical evaluation, broad spectrum antibiotics, pain control and IVF

Critical documentation

Serial VS, hernia presentation (reducible, incarcerated, strangulated), consultation.

Disposition

Admit patients with irreducible or strangulated hernia.

105 GI foreign body

Foreign bodies (FB) may be ingested or inserted into the anus. Most cases are accidental ingestion by pre-school children (peak three years) or adults with intellectual impairment or psychiatric conditions. GI FB may cause obstruction, perforation or infection. Risk is increased for objects that are large, sharp or that contain harmful substances (i.e. batteries). Large oesophageal foreign bodies may lead to airway compromise.

The first five minutes

- ABC, VS, glucose
- Initial focus is evaluation of airway

History and physical examination

Key historical features

- FB type, size, when ingested. Ask whether ingestion is oral or anal; ask specifically about toxidromes, corrosives and sharp objects
- Ask about symptoms: dysphagia, odynophagia, inability to swallow, coughing, drooling, pain and vomiting. Look for features of bowel obstruction, perforation, infection
- Existing GIT abnormalities (i.e. strictures) or previous surgery or trauma
- Intentional ingestion: assess mental state and suicide risk

Signs and symptoms

- For oral ingestions – assess for evidence of airway compromise, haematemesis, inability to swallow
- For any FB – assess for evidence of obstruction, perforation or peritonism.
- For rectal, examine for trauma and bleeding

Investigations

- Imaging: CXR/AXR (will only identify radio-opaque FB; may reveal pulmonary aspiration, mediastinitis, bowel perforation or obstruction; note size, shape and location of FB) ◇; CT scan without contrast ◈
- Endoscopy ◈: visualise object, assist in removal and evaluate for injury caused by the FB
- Metal detectors ◇: accurate and radiation free for localisation

Management

The goal of acute management is to determining whether disimpaction is needed or removal is indicated and to manage complications such as perforation, obstruction or infection.

Oesophageal FB

- Small objects in patients who can swallow, appear well and have no evidence of complications can be managed expectantly
- Urgent removal or surgical referral if: inability to swallow, evidence of perforation, evidence of mediastinitis, sharp objects, objects > 2 cm in diameter, all button batteries, airway compromise, FB at level of cricopharyngeus in a child, FB present for > 24 hours
- Removal should be by endoscope. If unavailable, consider removal with a Foley catheter for proximal objects that are not sharp. Pass catheter and inflate balloon distal to object. Lateral positioning essential to avoid displacing FB into airway
- Oesophageal food bolus impaction: limited evidence for glucagon 1–2 mg IV (adults – 0.03 mg/kg paeds (repeated once in 10 min if needed)) ◈. Meat boluses have a higher risk of perforation: rigid or flexible oesophagoscopy recommended ◈

Gastric, small or large intestinal FB

Indication for removal are complications, harmful substances (i.e. batteries) or size > 5 cm in length or > 2 cm in diameter. Surgical intervention may be required if endoscopic removal unsuccessful. ◈

Specific management

Batteries:
- Oesophageal impaction: immediate endoscopic removal with observation for perforation, fistula ◈
- All other cases: removal is usually indicated

Magnets:
- Two or more ingested magnets can attract each other across bowel wall, and lead to perforation. Refer for urgent

removal

Sharp objects (needles, toothpicks, safety pins, fish bones):
• In the oesophagus or stomach, endoscopic removal ◈
• If past the stomach, close observation and serial AXR ◇. Surgery if no progression in three days or if patient becomes symptomatic

Drug packer/stuffer: high mortality if leakage or rupture occurs:
• Diagnosis with CT or AXR; surgery if packets fail to progress. (See 🕮 Approach to acute poisoning, p. 652.)

Critical documentation

Airway status; investigation findings, suspected FB.

Disposition

> 80% FBs will pass spontaneously (examination of stool unnecessary). Admit high-risk or cases with complications to surgery.

106 Intussusception

Intussusception is a form of bowel obstruction caused by the involution of one segment of bowel into another. It is most common at the ileo-caecal junction and usually idiopathic in children. Children classically present at three months to six years; most are < 1 year with peak at 4–6 months. In adults, the cause is pathologic in up to 90% of cases, usually representing an occult oncologic process.

The first five minutes

As needed – ABC, VS, O_2, IV, cardiac monitor

History and physical examination

Key historical features

Child

• Crying: typically sudden onset intermittent crying and drawing up of the legs in young infants, or recurrent abdominal pain with periods of relief of pain between episodes
• Other features: bilious vomiting and currant-jelly appearing stools or blood per rectum are late findings. May also present with lethargy or AMS rather than as an abdominal process
• Ask about risk factors: increased risk in patients with known bowel disease: HSP or HUS due to bowel haematomas, cystic fibrosis due to thick stool, caeliac disease, Crohn's disease, lymphoma, or bowel infection (e.g. *Ascaris*) as a result of inflammation

Adults

• Other episodes or previous bowel obstruction
• Abdominal pain: typically intermittent, cramping, with vomiting and tenderness
• Ask about risk factors such as a concurrent cancer diagnosis

Signs and symptoms

• Signs of bowel necrosis such as toxic appearance (tachycardia, abdominal distension, bilious vomiting, progressive lethargy)
• Detailed examination of the abdomen, pelvis and groin:
  » Full abdominal exam: classically a sausage-shaped mass felt in the right side of the abdomen (especially in

children). Evaluate for signs of peritonitis

Differential diagnosis

Volvulus, hernia, trauma, tumours (benign or malignant), adhesions, ileus, viral or bacterial gastrointestinal infections.

Investigations

Diagnosis may be made on clinical suspicion in children.

- Imaging: AXR (perforation, 'target sign' (often over the right kidney), 'crescent sign' (soft tissue density projecting into the gas of the large bowel), obscured liver margin, no air in the caecum), US (bull's eye or coiled spring; Doppler can identify ischaemia) ◇; CT (may show intussusception but is not necessary unless attempting to characterise a mass, or if the diagnosis is unclear using other means) ◈

Management

The goal of management is to maintain haemodynamic stability, prevent/treat secondary complications such as peritonitis and reduce the intussuscepted bowel.

Non-surgical management

- Some resolve on their own if they are located in the small bowel and are short (< 3.5 cm)
- For infants with a typical presentation, it is possible to proceed directly to non-operative reduction with air enema or contrast enema reduction with fluoroscopy, this should be performed at a location with a surgeon available due to the risk of perforation ◇
- Little evidence for pre-medication with antibiotics to cover bowel organisms

Surgical management

- Small bowel intussusceptions, those > 7.5 cm, patients with symptoms lasting more than two days, bilious vomiting, fever or blood in stools are less likely to resolve with non-operative means
- Surgical management is required if there is evidence of perforation (peritoneal signs on exam or free air on AXR), or mass on imaging
- Keep all patients NPO, start broad spectrum antibiotics and provide pain control

Critical documentation

Suspected location and aetiology should be documented to support management decisions. In infants, recent rotavirus vaccine should be noted and reported.

Disposition

Observe non-surgically managed children for several hours (10% reoccurrence). Admit unstable patients or surgical candidates.

107 Hypertrophic pyloric stenosis

Hypertrophy and hyperplasia of smooth muscle at the pylorus leads to progressive gastric outlet obstruction. The condition is more common in males (5:1) and usually presents before 12 weeks of age. It is the most common cause of GI obstruction in infants.

The primary concern is progressive vomiting and poor oral intake leading to dehydration, electrolyte and metabolic abnormalities, hypoglycaemia and eventually total GIT obstruction.

The first five minutes

- As needed – ABC, glucose, O_2, IV, monitor

- Pay particular initial attention to dehydration, shock, hypoglycaemia and AMS. Correct hypovolaemia with IVF

History and physical examination

Key historical features

- Classically parents report vomiting immediately after feeding in a child who remains hungry. The vomiting is often described as ‘projectile’. May be blood tinged
- Ask about associated features: failure to thrive (ask about birth weight, current weight), dehydration, lethargy

Signs and symptoms

Evaluate the patient:

- May appear normal, hungry, or might have signs of dehydration, shock or jaundice (< 10% patients). May also present with lethargy or seizures
- Detailed examination of the abdomen:
  - » A palpable ‘olive’ or small mass in the right upper or middle quadrant at the lateral margin of the right rectus muscle just below the liver edge
  - » Peristaltic waves moving from left to right may be seen in the left upper quadrant after feeding

Differential diagnosis

Any other cause of persistent vomiting and/or bowel obstruction in an infant. Gastro-esophageal reflux disease, or volvulus with or without malrotation.

Investigations

This is a clinical diagnosis. Labs are required to identify electrolyte abnormalities: Children often present with a hypochloraemic hypokalaemic metabolic alkalosis.

Management

The goal of management is to provide resuscitation and correct electrolyte abnormalities.

- If not severely dehydrated maintain hydration with a paediatric dextrose containing fluid such as ½ DD (see 🕮 Paediatric resuscitation p. 18)
- If dehydrated, consider rehydration with a solution with a higher sodium content, or at faster rates (however, be careful not to replete sodium too quickly as may cause seizures)
- Definitive management is surgical repair after fluid and metabolic resuscitation

Critical documentation

Age, history and exam finding, investigations, management, and consultation communication.

Disposition

Admit all patients.

108 Approach to the child with jaundice

Jaundice is a yellow discoloration of the skin and sclera caused by hyperbilirubinaemia. A mild to moderate degree of jaundice in the newborn is expected, but large levels of unconjugated hyperbilirubinemia can cause irreversible toxic effects on the CNS (kernicterus).

The first five minutes

- As needed – ABC, VS, O_2, IV, glucose

• If extremely jaundice, seizures or poor skin turgor IVF bolus 20 ml/kg

History and physical examination

Key historical features

• Onset of symptoms (jaundice in the first 24 hours or after the third day are probably pathologic)
• Associated signs: poor feeding, lethargy, fever (sepsis can manifest as jaundice)
• Family history: sibling with jaundice, Gilbert's syndrome, history of anaemia
• Pregnancy history: mother's Rh status, drugs taken, viral illnesses, birth trauma, premature or low birth weight child

Signs and symptoms

• Look for signs of severe neurological dysfunction: lethargy/irritability, muscle tone, seizure, altered cry
• Overall appearance: jaundice first appears on face/ forehead then progresses to trunk and limbs:
  » Pressure on the skin can reveal underlying colour but may be difficult in darkly pigmented children
  » Look for cephalohaematoma, excessive bruising, petechiae
• Detailed examination of the abdomen and pelvis for hepatosplenomegaly

Differential diagnosis

Unconjugated

• Physiologic: normal increase in unconjugated hyperbilirubinemia due to immature enzymatic action and increased bilirubin
• Breast milk jaundice: increased enterohepatic reuptake of bilirubin
• Increased haemolysis: sickle cell, thallasaemia, G6PD deficiency, drugs
• Miscellaneous: hypothyroidism, Down's syndrome, polycythemia, sepsis, high intestinal obstruction

Conjugated

Always pathologic and include Dubin-Johnson syndrome, biliary atresia, congenital bile duct anomalies, cholelithiasis, primary sclerosing cholangitis, hepatitis, drug induced, infectious cholangitis.

Investigations

Diagnosis is clinical. Tests help determine the cause.
• Perform serum bilirubin (direct and indirect) ⟐:
  » If elevated direct bilirubin: LFTs, US (if conjugated hyperbilirubinemia, to define aetiology) ◇; viral hepatitis testing, serum ceruloplasmin, autoantibodies, liver biopsy ⟐
  » If elevated indirect bilirubin: CBC, type and cross, blood smear ◇; reticulocyte count, Coomb's test, thyroid function tests, syphillis serology, G6PD screen ⟐
• Blood, urine and CSF cultures (if bacterial infection is considered) ◇

Management

The goal of management is to prevent secondary neurological sequelae from hyperbilirubinemia and to identify and treat the underlying cause.
• Standard septic workup and early antibiotics for all with signs of infection.
• Any jaundice in a one day old neonate should be treated with immediate phototherapy without waiting for bilirubin levels
• Phototherapy or exchange transfusion is indicated based on the measured bilirubin level (see WHO guidelines below)

Table 108.1 Treatment of hyperbilirubinemia based on serum bilirubin level

	Phototherapy (mg/dL)	Exchange transfusion (mg/dL)
Day 1	Any visible jaundice	15
Day 2	15	25
Day 3	18	30
Day 4 and thereafter	20	35

- All patients with jaundice should be evaluated for concurrent infections
- Breastfeeding should be encouraged for all jaundiced children

Critical documentation

Clinical findings including hydration status, neurologic exam, abdominal exam with frequent reassessments.

Disposition

Admit patients with cholestatic jaundice, if unwell or if requiring phototherapy or exchange transfusion.

109 Approach to the adult with jaundice

Jaundice is a yellow discoloration of the skin and mucous membranes caused by hyperbilirubinemia. It becomes visible when the bilirubin level is above 2–3 mg/dL (34–51 μmol/l). It may be caused by over-production (pre-hepatic – haemolysis), inadequate metabolism (hepatic – drugs, infections, metabolic disorders), or inadequate excretion (post-hepatic – biliary obstruction) of bilirubin. Over-production and inadequate metabolism give rise to unconjugated hyperbilirubinemia; inadequate excretion (cholestatic jaundice) results in conjugated hyperbilirubinemia.

Hyperbilirubinemia rarely causes significant problems (with the important exception of kernicterus in babies (see p. 262) but may indicate a life threatening underlying cause.

The first five minutes

- ABC, VS, O_2, IV, glucose
- Pay particular attention to hypoglycaemia, evidence of liver failure (encephalopathy, bleeding), and features of sepsis

History and physical examination

Clinical evaluation aims to confirm the jaundice, identify dangerous associated conditions (sepsis, liver failure, hypoglycaemia) and find any potentially dangerous underlying cause (malignancy, severe haemolysis, severe hepatitis etc.).

Key historical features

Ask about onset, duration and severity of symptoms; discoloration of urine or stool (dark urine and pale stool = cholestatic jaundice). A prodrome of malaise may suggest viral hepatitis. Associated symptoms (weight loss, anorexia, fever, abnormal bleeding, features of encephalopathy, nausea, pruritis etc.). Recent travel and other friends or family members with similar symptoms.

Always document co-morbid diseases, medications and allergies. Specifically ask about the use of hepatotoxic medications (TB and HIV treatment among many others) and the use of traditional or unconventional therapies.

Signs and symptoms

- VS (fever, tachycardia) and general examination (mental state, severity of jaundice, evidence of bleeding, anaemia)
- Complete abdominal examination (look for hepatomegaly, liver or abdominal masses, gall bladder masses etc.)
- Search for features of chronic liver disease (anaemia, spider angiomata, superficial venous varicosities, cirrhotic

liver, splenomegaly, spider angiomata, ascites, oedema) and acute liver failure (AMS, asterixis, abnormal bruising or bleeding, hypoglycaemia, tachycardia)

Differential diagnosis and potential causes

Cause of jaundice may be suggested by the following (see Table 109.1):

- Acute jaundice in young and healthy – acute viral hepatitis (particularly when a viral prodrome, risk factors, or both); paracetamol overdose (see 📖 Paracetamol poisoning, p. 655)
- Acute jaundice after drug or toxin exposure in healthy patients – due to that substance
- A long history of heavy alcohol use – alcoholic liver disease (particularly with typical stigmata)
- A personal or family history of recurrent, mild jaundice without findings of hepatobiliary dysfunction – hereditary disorder (usually Gilbert syndrome)
- Gradual onset of jaundice with pruritus, weight loss, and clay-coloured stools – intrahepatic or extrahepatic cholestasis
- Painless jaundice in elderly patients with weight loss and a mass – biliary obstruction (cancer) until proven otherwise

Investigations

- Labs: CBC, electrolytes, renal, glucose, urinalysis, pregnancy test, LFT ◇; PT/PTT, viral hepatitis serology testing ◈
- Measure total and direct bilirubin, aminotransferase, and alkaline phosphatase levels in all patients. Results help differentiate cholestasis from hepatocellular dysfunction (see Table 109.1)
- Imaging: US (identify biliary obstruction; may also reveal liver masses or other pathology) ◇

Management

The goal of acute management is to identify and treat life threatening causes.

General management includes symptomatic treatment, avoidance of hepatotoxic drugs and possibly multivitamins. Pay careful attention to glucose, fluid and electrolyte management.

Ascending cholangitis

- Patients often have Charcot’s triad: fever, jaundice, and RUQ pain
- Investigation – US (obstructed biliary tree)
- Management – IVF, broad spectrum antibiotics including gram-negative cover (e.g. ciprofloxacin and metronidazole, ERCP) ◈

Acute alcoholic hepatitis

- When of sufficient severity to cause jaundice, hepatic encephalopathy, or coagulopathy, mortality can be substantial. Sub-acute onset of fever, hepatomegaly, leukocytosis, marked impairment of liver function, and manifestations of portal hypertension (e.g. ascites, hepatic encephalopathy, variceal haemorrhage)
- Rapid assessment is essential. Identifying sources of infection, excluding bacterial peritonitis and gastrointestinal bleeding, evaluating fluid status, and excluding electrolyte abnormalities, as well as recognising malnutrition, are important issues to consider during the initial assessment
- Management: nutritional support, adequate protein and caloric intake, treat co-existing infections, correct electrolyte abnormalities

Critical documentation

Location and examination characteristics (tenderness, tympany, mobility, etc.), key history, investigation results, treatment plan.

Disposition

Admit ill-appearing patients and those with hypoglycaemia, abnormal bleeding, sepsis, evidence of liver failure, conjugated hyperbilirubinaemia or sonographic evidence of biliary obstruction.

Discharge and follow-up well patients with unconjugated hyperbilirubinemia, no evidence of haemolysis or complications and (if US available) no US evidence of biliary obstruction.

Table 109.1 Differential diagnosis and potential causes of jaundice in adults

Mechanism	Examples	Suggestive findings
Unconjugated hyperbilirubinemia		
Increased bilirubin production	Haemolysis Ineffective erythropoiesis	Falling Hgb, dark urine, rapid onset ±splenomegaly
Decreased hepatic bilirubin uptake	Heart failure Infection	See 🕮 CCF p. 104 See 🕮 Toxicology p. 652
Decreased hepatic conjugation	Gilbert syndrome Ethinyl estradiol/ hyperthyroidism	
Conjugated hyperbilirubinemia (typically a mixed conjugated/unconjugated pattern)		
Hepatocellular dysfunction	Drugs, toxins, viral hepatitis Alcoholic liver disease, haemochromatosis, primary biliary cirrhosis, primary sclerosing cholangitis, Wilson disease	Aminotransferase levels usually > 500 U/L
Intrahepatic cholestasis	Alcoholic liver disease, drugs, toxins, viral hepatitis, TB Less infiltrative disorders (e.g. amyloidosis, lymphoma, sarcoidosis), pregnancy, primary biliary cirrhosis	If severe, clay-coloured stools, steatorrhoea If long-standing, weight loss Alkaline phosphatase and GGT usually > 3 times normal Aminotransferase levels < 200 U/L
Extrahepatic cholestasis	Common bile duct stone, pancreatic cancer Acute cholangitis, pancreatic pseudocyst, primary sclerosing cholangitis, common duct strictures	Depending on cause, manifestations similar to those of intrahepatic cholestasis or a more acute disorder (e.g. abdominal pain or vomiting due to a common bile duct stone or acute pancreatitis) Alkaline phosphatase and GGT usually > 3 times normal Aminotransferase levels < 200 U/L

110 Mesenteric ischaemia

Acute mesenteric ischaemia results from inadequate blood supply to the intestine. It may be caused by arterial or venous occlusion or low flow states. The cardinal features are abdominal pain with a paucity of physical findings in older patients (typically > 50) with vascular risk factors or disease. Diagnosis can be extremely challenging and untreated cases are associated with high mortality (> 60%).

The first five minutes

ABC, VS, O_2, IV, glucose, cardiac monitor

History and physical examination

Key historical features

• Abdominal pain: typically acute and severe, sudden onset (arterial embolism) vs indolent (venous occlusion)
• Associated features: nausea, vomiting, initial diarrhoea, blood in the stool and eventually features of sepsis
• Factors: vascular risk factors/disease and previous similar episodes

Signs and symptoms

• Signs of bowel necrosis, pain and tachycardia, abdominal distension, bilious vomiting, progressive lethargy

• Full abdominal examination: may be benign even in severe disease, pain is typically out of proportion to tenderness. Evaluate for signs of peritonitis (late, poor prognosis)

History and physical examination vary with type of ischaemia (see Table 110.1).

Table 110.1 Type of ischaemia and history and physical examination

Type	Historical features	Risk factors	Signs and symptoms
Arterial embolism	Common; male predominance. Classical presentation: sudden, severe pain associated with gut emptying (diarrhoea, vomiting)	Cardiac disease (atrial fibrillation), aortic disease, or iatrogenic (recent arterial instrumentation)	Hallmark: pain out of proportion to physical exam Examination consistent with peritonitis is a late sign
Arterial thrombosis	Women > men, most commonly > 60 years Association with post-prandial pain, food avoidance, and weight loss	History of atherosclerosis (or risk factors)	Dull abdominal pain
Non-occlusive mesenteric ischaemia	Abdominal pain may be absent or variable	Associated with vaspressors, digoxin and dialysis	Abdominal distension, fever, gastrointestinal bleeding in a critically ill patient may be the only sign
Mesenteric vein thrombosis	Least common form; presents acutely (< 4 weeks) and chronically (> 4 weeks) Various presentations from acute abdominal pain to vague abdominal discomfort with nausea/vomiting, and/or diarrhoea	Associated with personal or family history of lower extremity blood clot, hypercoaguable states (i.e. Factor V Leiden, protein C & S deficiency), malignancy, OCP, portal hypertension, sepsis, or post-operative	Abdominal exam may be unremarkable or with concerning peritoneal signs

Differential diagnosis

Any cause of abdominal pain: perforated ulcer, bowel obstruction, AAA, aortic dissection, diverticulitis, gastroenteritis.

Investigations

Definitive diagnosis depends on evaluation of the mesenteric vasculature (angiography, CT or MRI angiography, or Doppler US) ◈. Other investigations do not contribute to finding the cause but may help to identify complications (acidosis, renal failure, electrolyte abnormalities) or alternative diagnoses (pancreatitis).

• Labs: CBC (leukocytosis), electrolytes, renal, ABG (metabolic acidosis – late) ◇; lactate (raised), PT/PTT (if abnormal coagulation suspected) ◈

• Imaging: AXR ◇ (free air, pneumatosis, portal venous air and ileus)

Management

The goal of acute management is analgesia, fluid resuscitation and rapid access to definitive treatment.

• Aggressive IVF; broad spectrum antibiotics; analgesia, and anticoagulation (i.e. heparin infusion if available)

• Definitive treatment with intra-arterial thrombolysis or laparotomy with vascular repair and resection of necrotic bowel

Critical documentation

Document clinical findings, frequent reassessments, response to therapy, expert consultation, and imaging results.

Disposition

Admit all patients to surgery, preferably to ICU ◈.

111 Acute pancreatitis

Pancreatitis is inflammation and auto digestion of the pancreas. Intense local (and eventually systemic) inflammation results in pain and local (tissue destruction, abscess formation) and systemic complications (DIC, metabolic derangements, shock).

The two most common causes are alcoholic pancreatitis and gallstone disease. Other rare causes include viral infection, trauma, hyperlipidaemia, hypercalcaemia and more. For patients with necrotising pancreatitis, the mortality rate approaches 50%.

The first five minutes

- ABC, VS, O_2, IVF, cardiac monitor, analgesia

History and physical examination

Key historical features

- Abdominal pain: patients classically present with dull, boring epigastric pain and intractable nausea and vomiting
- Risk factors: history of alcoholism, gallstones, recent infection (mumps, coxsackie virus, HIV and typhoid), hyperlipidaemia, hyperparathyroidism with hypercalcaemia, autoimmune disorders, recent ERCP, and trauma. Medications: azathioprine, oestrogens, thiazide diuretics, NSAIDS, corticosteroids, antibiotics, furosemide, valproic acid, risperidone, proton pump inhibitors

Signs and symptoms

- Signs of bowel necrosis (tachycardia, abdominal distension, bilious vomiting, progressive lethargy)
- Signs of infection: fever and hypotension
- Signs of severe disease: hypotension (bleeding, dehydration, sepsis, third spacing or myocardial depression), tachypnoea (hypoxia, atelectasis, pleural effusion or ARDS). May be restless, anxious or depressed. Haemorrhagic pancreatitis: bruising of flanks (Grey Turner's Sign), or periumbilical ecchymosis (Cullen's Sign)
- Detailed examination of the abdomen:
  - » Tenderness in left upper quadrant and/or epigastric area, decreased bowel sounds, distended and tympanic abdomen

Differential diagnosis

Any cause of upper abdominal pain, particularly complications of peptic ulcer disease. Also consider: gallbladder disease, peritonitis, bowel obstruction, hollow viscus perforation, mesenteric ischaemia, aortic aneurysm, ACS etc.

Investigation

Tests should aim to confirm the diagnosis (lipase or amylase – lipase is a superior test; do not do both) and determine severity/complications (renal function, electrolytes, Ca, etc.)

Imaging is rarely needed unless severe disease or local complications are suspected.

- Labs: CBC (anaemia, white count response), electrolytes, renal, LFTs (ALT > 80 indicative of biliary pancreatitis (gallstone that passes through the bile duct and temporarily lodges at the sphincter of Oddi)) ◇; amylase (> 3 × normal) or lipase (> 2–3 × normal; high specificity) (absolute levels are not indicative of severity) ⟐
- Imaging: AXR (bowel obstruction, ileus), CXR (free air, atelectasis, pneumonia pleural effusion) ◇; contrast CT abdomen (severe disease or concern for pseudocyst, haemorrhage, abscess and necrotising pancreatitis) ⟐

Management

The goal of acute management is analgesia, supportive care, and detection and management of complications.

- NPO except for mild cases, but early nutritional support improves outcomes

- IV analgesia, IV antiemetics
- IVF to maintain urine output (via catheter) of 0.5–1 ml/kg/hr
- NGT for intractable vomiting ◇
- Surgical consultation for biliary pancreatitis, severe pancreatitis or if local complications are found
- Consider broad spectrum IV antibiotics if suspicion of infective complications

Critical documentation

Document detailed history and physical, VS, frequent reassessments, response to therapy, expert consultation, and imaging results.

Disposition

- Admit: moderate to severe illness i.e. those with three or more of Ranson's criteria on admission (indicates high mortality risk): age > 55, WBC >16 000, glucose > 10 mmol/L, AST > 250 U/l, LDH > 350 IU/l
- Admit to ICU: septic, critically ill, persistent hypotension
- Discharge: mild cases, tolerating oral liquids

112 Peptic ulcer disease

Mucosal ulceration of the gastric or duodenal mucosa caused by imbalance between mucosal protective function and the damaging effects of gastric secretions, GIT enzymes and inflammation. Most cases are associated with *H. pylori* infection or over-use of aspirin or NSAIDs. Other important associations include smoking, alcohol abuse and GIT malignancy.

Patients usually present with pain and/or dyspepsia, but may also present with features of important complications such as GIT bleeding, perforation or obstruction. See 🕮 RAP GI bleeding, p. 66 and Upper GI bleeding, p. 246.

The first five minutes

- As needed – ABC, VS, IVF, ECG monitor
- If patient presents with shock, peritonitis or severe bleeding, start immediate resuscitation with IVF bolus followed by blood products

History and physical examination

Key historical features

- Abdominal pain: symptoms often related to food and relieved by antacids
- Features that may suggest the cause: NSAID use, smoking, alcohol abuse, features of malignancy (early satiety, weight loss)
- Associated features: fullness, bloating, vomiting up to several hours after a meal (obstruction), sudden onset of severe sharp abdominal pain with signs of peritonism and fever (perforation). Haematemesis, melaena, symptomatic anaemia, shock (bleeding)

Signs and symptoms

- Look for evidence of blood loss (anaemia, shock), perforation (peritonitis, fever) and cancer (weight loss, abdominal masses etc.)
- Detailed examination of the abdomen and rectum
- Tenderness in the epigastrium. Distension and vomiting may indicate obstruction. Perform a rectal exam to look for bleeding/melaena

Differential diagnosis

- Any cause of upper abdominal pain, dyspepsia and peritonitis
- Gastritis, drug induced dyspepsia (NSAIDs, aspirin, theophylline, digitalis), gastric/duodenal carcinomas,

infections (gastric/duodenal TB with ulceration), granulomatous diseases, Crohn's disease, Zollinger-Ellison syndrome, functional dyspepsia/non-ulcer dyspepsia

Investigations

Important initial investigations include Hgb and erect CXR (free air under the diaphragm indicating perforation) ◇. Further testing depends on the clinical scenario. Definitive diagnosis (and often treatment) can be made with endoscopy.

Also consider *H. pylori* testing ◈ for patient < 55 years. Serum enzyme-linked immunosorbent assay – most rapid, least sensitive (89%); stool antigen test – inconvenient but more accurate; Urea breath test – significantly more expensive.

Management

The goal of acute management is symptom relief and detection and management of complications, especially bleeding.

- High index of suspicion (or *H. pylori* positive) and no imaging:
  - » Eradicate *H. pylori* (proton pump inhibitor and amoxicillin and clarithromycin OR metronidazole – 14 days)
  - » Antisecretory agent (proton pump inhibitor, H2 blocker) for 4–8 weeks
  - » Discontinue NSAIDS, smoking, alcohol, illicit drug use
- Imaging reveals ulcer → biopsy ulcer, eradicate *H. pylori*, antisecretory medication
- Imaging reveals no ulcer → antisecretory trial, follow clinically as outpatient
- Immediate emergency surgery if perforation or failure to achieve haemostasis

Critical documentation

Serial VS, frequent reassessments, response to therapy, expert consultation, and test results.

Disposition

- Admit patients with evidence of acute complications (severe anaemia, shock, peritonitis) to surgery
- Discharge other patients with eradication therapy, lifestyle advice and follow-up; patients at risk of malignancy (older, alcohol/tobacco use, weight loss, masses, early satiety) require urgent outpatient investigation

113 Vomiting

Vomiting is a frequent presenting complaint and may be caused by GIT, CNS and cardiac conditions. Vomiting may have serious consequences (hypovolaemia, acid-base abnormalities, electrolyte abnormalities, oesophageal injury, and pulmonary aspiration) and may be due to a serious underlying disease (GIT malignancy, meningitis, some toxidromes, DKA, AMI, mesenteric ischaemia, sepsis, etc.). The most important considerations during the initial encounter are recognition of serious aetiologies, fluid loss, and electrolyte derangement.

The first five minutes

- ABC, IV, glucose, cardiac monitor ◇

History and physical examination

Key historical features

- Onset (abrupt or insidious), timing (when fasting, during or directly after eating, continuous, irregular), character of emesis (bilious, bloody)
- Bowel changes, fever, AMS, headache, weakness, tetany, spasm or seizures, haematemesis, melaena, abdominal pain, contacts with similar symptoms. In children ask about weight loss, last wet diaper, making tears
- Pre-existing medical conditions, medication and allergies. Recent travel. Possible toxic exposures

Signs and symptoms

• Assess for signs of dehydration and/or shock
• Neurologic exam: papilloedema, motor deficits, stiff neck
• Abdomen: localised tenderness, guarding, rebound, hyper/hypoactive bowel sounds, abnormal masses, hepatosplenomegaly

Differential diagnosis

• CNS: ICH, meningitis, increased intracranial pressure (ICP), migraine, seizure, vestibular disorder
• GI: obstruction, appendicitis, cholecystitis, cholangitis, hepatitis, pancreatitis, perforation, mesenteric ischaemia, PUD, peritonitis
• Infectious: AGE (bacteria/toxins), pneumonia, adenovirus, Norwalk, rotavirus, SBP, UTI, pyelonephritis
• Medications: antiarrhythmics, antibiotics, anticonvulsants, chemotherapeutics, digoxin, NSAIDs, opiates, overdose/withdrawal, radiation
• Toxins: organophosphates, arsenic, ricin, ethanol
• Metabolic: diabetic ketoacidosis, adrenal disorder, uraemia, thyroid disorder, pregnancy (hyperemesis gravidarum), parathyroid disorder
• Miscellaneous: acute coronary syndrome, acute glaucoma, nephrolithiasis, pain, psychiatric disorder, abnormal electrolytes, post-tussive emesis
• In addition to the above, in children consider: pyloric stenosis, malrotation with or without volvulus, intussusception, Hirschsprung’s disease, intestinal atresia, adrenal insufficiency or crisis, inborn errors of metabolism, GORD

Investigations

Diagnosis is often made via clinical examination; always consider pregnancy in women of appropriate age. Consider any other investigation directed to the underlying cause.

• Labs: electrolytes, renal (signs of severe dehydration or shock), glucose, ABG ◇
• ECG: ◇ (evidence of electrolyte abnormalities or ischaemia)
• Imaging: erect CXR or AXR (bowel perforation or obstruction) ◇

Management

The goal of acute management is symptom relief and correction of fluid and electrolyte abnormalities. Find and treat any possible dangerous cause.

Dehydration

• Mild to moderate dehydration, ORS by mouth or NGT (according to WHO guidelines)
• If severe dehydration/shock, bolus 20 ml/kg of 0.9% NS with two further boluses if further signs of shock (see 🕮 Shock, p. 88)
• Monitor the response to therapy by frequent repeat clinical assessment
• Note that guidelines differ for the malnourished child (see 🕮 SAM p. 394)
• Encourage breastfeeding as tolerated for children

Medications

Anti-emetics (metoclopramide, prochlorperazine, promethazine, ondansetron).

Critical documentation

Serial VS, hydration status, mental status, serum K, response to IVF.

Disposition

Admit patients unable to take PO, with severe or symptomatic hypokalaemia, severe dehydration, evidence of sepsis

or malnourished.

114 Oesophageal emergencies

Oesophageal emergencies include perforation, bleeding, obstruction/impaction, ingestion of caustic material or foreign body, and inflammation.

The first five minutes

- ABC, VS, IVF, monitors – as needed
- Perforation, bleeding or caustic ingestion may require immediate, aggressive supportive care

History and physical examination

Key historical features

Common complaints include dysphagia or odynophagia.

- Oesophageal perforation/rupture: chest pain following violent retching/vomiting (Boerhaave’s syndrome), recent endoscopy
- Obstruction/impaction: dysphagia. Time course (progressive favours neoplasm; sudden onset favours ring/stricture/diverticulum), progression, perceived area of obstruction important for localisation
- Ingestion: time course, material ingested
- Oesophagitis: odynophagia ± foreign body sensation

Signs and symptoms

- Perforation/rupture: fever, crepitus, pleural effusion
- Obstruction/impaction, ingestion: evaluate for airway compromise
- Oesophagitis: white plaques in pharynx associated with *Candida*.

Differential diagnosis

Odynophagia

ACS, PTX, PE, oesophageal rupture/perforation (iatrogenic, foreign body, trauma-related), oesophagitis, pneumonia, pneumomediastinum/mediastinitis, diffuse oesophageal spasm, achalasia, peritonsillar abscess.

Dysphagia/obstruction

Oropharyngeal dysphagia/neuromuscular disorder, neoplasm, GORD, oesophageal diverticula, oesophageal ring/stricture.

Investigations

Most diagnoses are made on history and physical examination.

- Foreign body ingestion: CXR and lateral soft tissue neck XR ◇ (to locate)
- Perforation/rupture: CXR, AXR, neck XR ◇ (free air, pneumomediastinum, PTX, pleural effusion)
- Dysphagia/obstruction: barium oesophagram ◇ (to delineate anatomy)
- Odynophagia: contrast oesophagography ◈ (water soluble contrast; 10% false negative rate; if negative, barium may have higher sensitivity for small perforations)

Management

Oesophageal perforation/rupture

• NPO, aggressive IVF resuscitation, antibiotics (for gram-positive, gram-negative and anaerobes)
• Chest tube ◇ for effusion or PTX
• Emergency surgical intervention

Obstruction/impaction

• Obstruction due to malignancy should have supportive care as needed, referral to oncologist/surgeon for further management as soon as possible
• Food impaction/foreign bodies (📖 p. 256)

Caustic ingestion

• Immediate evaluation of and management of airway and haemodynamic support. No benefit (and potential harm) to activated charcoal, irrigation, neutralisation
• Immediate surgical evaluation for evidence of perforation or mediatstinitis ◈

Oesophagitis

• Infectious: consider immunosuppression workup if due to *Candida* and treat with fluconazole◈. Other pathogens include CMV and HSV
• Inflammatory:
  » Acid suppressive therapy ◇ if reflux-associated (consider referral for endoscopy ◈ to evaluate for Barrett's oesophagus)
  » Remove offending agent if medication-related

Critical documentation

Document VS, frequent examinations, response to therapy, expert consultation.

Disposition

Discharge patients with uncomplicated oesophagitis. Admit to surgery if oesophageal rupture, button battery, sharp object ingestion.

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Part

4

G. Haematology and oncology

115 Sickle cell disease

A common disease with high morbidity/mortality. Leading causes of death in patients with this condition include acute chest syndrome and septicaemia.

Condition	History and Physical	Possible causes/ differential diagnosis	Investigations	Management
Vaso-occlusive crisis (VOC)	Pain in chest, back, hips, shoulders, proximal arms, legs Caused by increased sickling	Causes: infection temperature change dehydration trauma Differential: any syndrome causing pain	Hgb CBC ◇ Reticulocyte count ◈	Treat the precipitating cause. PO hydration Analgesia Consider opiates or ketamine ◇ O_2 (goal sats 95%) IVF (isotonic)
Acute chest syndrome	Infiltrate on CXR PLUS Cough or fever or tachypnoea or chest pain or wheezing	Cause: pulmonary infarction triggered by infection sequestration thrombo-embolism. Differential: fat embolism MI PE Pneumonia	Hgb CBC, blood cultures ◇ Reticulocyte count ◈ CXR ◇	Analgesia O_2 Antibiotics for pneumonia Blood transfusion to goal Hgb 10 if: saturation < 92% (room air), Hgb < 5, Hb drop > 20% from baseline, clinical worsening Exchange transfusion ◈ in severe disease
Fever	Fever, any systemic symptoms indicative of potential infection	Cause: sickle cell patients should be considered vulnerable to severe infection and septicaemia due to functional asplenia	Work-up based on history and physical Malaria testing Urinalysis CXR Blood cultures ◇ Urine culture ◇	Supportive care Broad-spectrum empiric antibiotics Empiric malaria treatment if testing not possible

Condition	History and Physical	Possible causes/ differential diagnosis	Investigations	Management
Splenic sequestration	Acute, painful enlargement of spleen with decrease in Hgb and platelets	Cause: sequestration of sickled RBCs in the spleen Differential: malaria thalassemia haemolytic anaemia	Hgb CBC ◇ Reticulocyte count ◈	Hydration Analgesia Blood ◇, or platelet ◈ transfusion to goal Hgb 7–8 and platelets > 20 000 Avoid over-transfusion due to risk of hyperviscosity syndrome
Aplastic crisis	Acute, severe drop in Hgb, white cells, platelets	Cause: infection (parvovirus) Folate deficiency Marrow necrosis	Hgb CBC ◇ Reticulocyte count ◈	Self-resolving Supportive care Exchange transfusion ◈ in severe cases
Stroke	Sudden onset of focal neurologic deficits	Cause: VOC crisis in the brain Differential: non-occlusive ischaemic or haemorrhagic stroke	Hgb CBC, Electrolytes ◇ Reticulocyte count ◈ Head CT ◈	Supportive care Exchange transfusion ◈
Priapism	Painful erection lasting > 4 hours	Cause: penile VOC	Hgb CBC ◇ Reticulocyte count ◈	Hydration Analgesia Therapeutic aspiration ◇ Intracavernous sympathomimetic ◈

Management

The goal of acute management is to decrease amount of sickling and frequency of VOCs.

- Nutrition:
  - » Folate deficiency: folic acid 1g PO daily
  - » Daily iron-free multivitamin
- Hydroxyurea ◇: reduces VOCs and hospitalisations. Requires close follow-up
- Blood transfusion ◇:
  - » Indicated for acute chest, acute stroke, acute symptomatic anaemia
  - » Transfuse for Hgb < 5 or > 20% drop from patient's known baseline
- Prophylactic antibiotics: prevent infection and mortality in children
  - » 3 months–3 years: penicillin V 125 mg PO twice per day
  - » 3–5 yrs: penicillin V 250 mg PO twice per day
  - » > 5 years: consider stopping
- IVF are essential for treatment of dehydration but must be used with caution in chest crisis. Monitor pulmonary exam and O_2 sat ◇

Critical documentation

Baseline Hgb, history of sickle cell complications.

Disposition

- Discharge uncomplicated VOC with adequate pain relief

- Admit VOC not responsive to analgesia, patients with fever or signs of septicaemia, acute chest, priapism, stroke, aplastic crisis, or splenic sequestration

116 Anaemia

WHO defines anaemia as haemoglobin < 13 g/dl in men and < 12 g/dl in women.

The first five minutes

VS, O_2, identification and control of active bleeding, identification and correction of high-output heart failure (📖 p. 104).

History and physical examination

Key historical features

Dyspnoea, fatigue, ↓ exercise tolerance, hyperdynamic state (bounding pulses, palpitations, 'roaring in the ears'); if severe – lethargy, confusion, sweating, cold extremities, postural dizziness, syncope.

Signs and symptoms

- Pale conjunctivae, palms and soles; tachycardia, tachypnoea (low Hgb)
- Hypoxia, crackles, tachycardia, tachypnoea, puffy feet or hands, oedema (high-output heart failure).
- Jaundice (haemolysis), splenomegaly (thalassemia, neoplasm, chronic haemolysis), petechiae/purpura (bleeding disorder), glossitis (iron, folate, vitamin B12 deficiency), koilonychias (iron deficiency), neurological abnormalities (vitamin B12 deficiency).

Possible causes and differential diagnosis

Reduced RBC production

- Nutritional deficiency – iron, B12, folic acid
- Depressed bone marrow function – malignancy, aplastic anaemia
- Infection – HIV, TB
- Erythropoeisis – chronic renal failure, hypothyroidism

RBC loss

- Acute or chronic haemorrhage – trauma, ulcer, hookworm

RBC destruction (haemolysis)

- Sickle cell disease, thalassemia, G6PD deficiency, malaria

Investigations

- Labs: point-of-care Hb; CBC with peripheral smear ◇
- Hb normal values:
  - » > 13 g/dl men, > 12 g/dl women, > 11 g/dl pregnant women
  - » > 11 g/dl children, > 9.5 g/dl 2 to 6 months, > 13.5 g/dl neonates
- Mean corpuscular volume (MCV): average volume of RBCs:
  - » Macrocytosis (MCV > 100): megaloblastic anaemia (B12, folate deficiencies), hypothyroidism, nucleoside reverse transcriptase inhibitors
  - » Microcytosis (MCV <76): iron deficiency, thalassemia, lead poisoning
- Reticulocyte index (RI):
  - » Normal 0.5–2% in healthy adults

» RI should be elevated, indicating appropriate bone marrow response to anaemia by making new RBCs (reticulocytes)

» 'Normal' RI in anaemia is inappropriately low and suggests hypoproliferative disorder

• Haemolysis markers:

» Elevated unconjugated bilirubin, elevated LDH, and low haptoglobin indicate RBC destruction

» A peripheral smear may show schistocytes

Low RI and normal bilirubin/LDH: hypoproliferative anaemia
High RI and high bilirubin/LDH: haemolytic anaemia
Low RI and high bilirubin/LDH: ineffective erythropoiesis
High RI and normal bilirubin/LDH: blood loss

Management

Blood transfusion

The goal of acute management is to increase O_2-carrying capacity of the blood, improving O_2 delivery to heart and brain. Patients with chronic anaemia with Hgb > 5 rarely require emergency transfusion.

Transfuse packed RBC (first preference) or whole blood if:

• Acute, severe anaemia with haemodynamic instability or ischaemia

• Hgb < 5 g/dl

» Hgb < 8 g/dl in patients with known ischaemic heart disease, ongoing bleeding, or if symptomatic

Iron deficiency

The goal of acute management is to increase iron stores and thereby RBC production.

• Dosing in elemental iron. Ferrous gluconate – 120 mg elemental iron/g, ferrous sulfate 200 mg elemental iron/g, ferrous fumarate 330 mg elemental iron/g. Effectiveness and side effects similar for all:

» Children: 2 mg/kg PO three times daily with food

» Adults: 50–100 mg PO three times daily with food

• Duration: three months to reverse anaemia, six months to replete iron stores

• If suspect nutritional deficiency rather than bleeding, supplement folic acid as well

Empiric treatment of helminthic infection

The goal of acute management is to decrease chronic intestinal blood loss.

See Parasitic infections of the GI tract (📖 p. 354) for treatment.

Folic acid deficiency

The goal of acute management is to correct nutritional deficiency.

• < 1 year: folate 0.5 mg/kg daily for 4 months

• > 1 year, adults: folate 5 mg daily for 4 months

B12 deficiency

The goal of acute management is to correct nutritional deficiency.

• 1 mg B12 IM daily for 1 week, then weekly for 4–8 weeks, then monthly for as long as anaemia persists

G6PD deficiency

• Supportive care during acute haemolysis

• Prevention. Avoid haemolytic precipitants: infection, diabetic ketoacidosis, fava beans, oxidative drugs (sulfas, dapsone, primaquine, nitrofurantoin)

Critical documentation

Symptoms, initial Hgb, baseline Hgb if known, treatment given and response.

Disposition

Discharge if stable with no signs of high-output failure or acute illness; re-check Hgb one month. Admit if unstable, unclear aetiology, significant ongoing bleeding, need for further work-up or requirement for multiple transfusions.

117 Coagulopathies

A number of bleeding disorders exist. The most severe is disseminated intravascular coagulopathy (DIC) – a syndrome in which there is inappropriate, widespread activation of coagulation and intravascular fibrin formation with concomitant breakdown of fibrin (fibrinolysis) and coagulation factor consumption. Mortality approaches 75%.

First five minutes

- ABC, VS, O_2, IV access, cardiac monitor ◇.
- Control haemorrhage, volume resuscitation. **Be mindful of aggressive fluids in severe, acute anaemia.** Obtain blood products stat from blood bank

History and physical examination

Key historical features

History of bleeding problems; medication use (aspirin, clopidogril, LMWH, warfarin); recent trauma, constitutional symptoms. Precipitating factors.

Signs and symptoms

- Assess **severity and duration of current bleeding diathesis**, including estimated blood loss, physical signs of severe **anaemia**, hypovolaemic **shock**

Patterns of disease:

- Platelets or vascular endothelium: epistaxis, superficial ecchymosis, purpura, prolonged bleeding after minor surgery, nasal or dental oozing
- Coagulation or fibrinolysis: haemarthrosis, deep ecchymoses, significant and prolonged bleeding, family history of bleeding
- Toxins: herbals, prescriptions, nonsteroidal anti-inflammatories, aspirin
- TTP: fever, AMS
- Thrombocytopaenia: splenomegaly
- Malignancy: lymphadenopathy
- Vitamin K deficiency: malnutrition
- HSP, HUS: recent diarrheal or upper respiratory infection
- HELLP, eclampsia: pregnancy
- Liver failure: current or remote alcohol use, ascites, stigmata of liver disease

DIC

DIC is a constellation of signs caused by many different illness states. Few patients in DIC present with signs of shock. Look for:

- **Bleeding**: easy bruising, petechiae, haemorrhage, bleeding from venopuncture sites
- **Thromboembolism**: focal ischaemia, limb swelling, dyspnoea
- **Multi organ failure**: renal, hepatic, respiratory, CNS

Possible causes and differential diagnosis

Possible causes

Thrombocytopaenia

- Sequestration: splenomegaly
- Destruction: ITP, TTP/HUS, HSP
- Drug-induced: H2-blockers, rifampicin, alcohol, oestrogens
- Malignancy: lymphoma, myelofibrosis, myeloma, myelodysplasia
- Bone marrow failure: aplastic anaemia
- Infection: TB, CMV, HIV, EBV, malaria, rickettsia, rubella, VHF, sepsis

Normal PT/PTT, elevated PTT

- Haemophilia, heparin, von Willebrand's, lupus anticoagulant

Elevated PT/PTT, normal PTT

- Warfarin, liver disease, vitamin K deficiency, malnutrition

Elevated PT/PTT, elevated PTT

- DIC (due to infection, sepsis, malignancy, trauma, obstetric emergencies, acute respiratory distress, chronic liver and pancreatic disease)

Differential diagnosis

Thrombotic microangiopathies (TTP/HUS)

- Labs: CBC (thrombocytopaenia; schistocytes on peripheral smear), renal (elevated urea and creatinine) ◇; LDH, indirect bilirubin (elevated) ⟐
- **Thrombotic thrombocytopaenic purpura** (TTP)
  - » Classic pentad: thrombocytopaenia, neurologic symptoms, fever, renal failure, red cell destruction; more prevalent in adults
- Haemolytic uraemic syndrome (HUS)
  - » More severe renal abnormalities; often preceded by dysentery; more prevalent in children

Henoch-Schönlein Purpura (HSP)

- Palpable purpura rash, arthritis, abdominal pain, gastrointestinal bleeding
- More prevalent in children, classically follows an upper respiratory infection
- Labs: CBC (platelets normal), renal (mildly elevated urea and creatinine), urinalysis (may have haematuria) ◇; PT/PTT (normal) ⟐

Idiopathic thrombocytopaenic purpura (ITP)

- Severe auto-immune thrombocytopaenia in absence of any other abnormality
- May be acute (usually post-infection in children) or chronic (usually adults). At risk for intracranial haemorrhage
- Labs: CBC (thrombocytopaenia) ◇

Haemophilia A and B

- X-linked inheritance. Often history of bleeding disorder in male family members
- Spontaneous bleeding into joints, muscles, and gastrointestinal tract. Intracranial haemorrhage with minor head trauma and spontaneous intracranial haemorrhage are major life threats
- Labs: PTT (elevated – as screening), factor VIII, factor IX assay (confirmatory) ⟐

von Willebrand disease

- Inherited. Often family history of bleeding disorder

- Mucocutaneous bleeding, including epistaxis, gingival bleeding
- Diagnosis primarily clinical, based on patient and family history

DIC

Severe, diffuse intravascular coagulation uses clotting factors, platelets, red cells, resulting in consumptive coagulopathy. Patients are critically ill. Differential includes hepatitis, cirrhosis, sepsis, other coagulopathies, vitamin K deficiency.
- Labs: CBC (schistocytes on peripheral smear) ◇; LDH (elevated); PT /PTT and D-dimer (elevated) ◈

Investigations

- Labs: CBC, electrolytes, renal, malaria testing, pregnancy test (for DIC) ◇; LDH, fractionated bilirubin, peripheral smear, bleeding time, D-dimer, fibrinogen, PT/PTT ◈

In DIC, further investigations should also be directed towards the possible cause.

Management

The goal of acute management is stabilisation of haemorrhage, correction of severe cytopaenia, and initiation of appropriate work-up.
- O_2, haemorrhage control, volume resuscitation
- Supportive care and treatment of underlying exacerbating condition
- Transfuse packed RBC if active haemorrhage, severe anaemia, signs of haemodynamic instability ◇
- Transfuse platelets for <10 000 in absence of bleeding < 50 000 if bleeding, < 100 000 if severe bleeding ◈

Condition-specific interventions

- **ITP**: high-dose steroids, immunoglobulin ◈, rarely splenectomy
- **TTP**: plasmapheresis, haemodialysis ◈
- **HUS**: haemodialysis ◈
  - » Transfuse platelets only in setting of active bleeding in HUS/TTP, as platelet transfusions may worsen the underlying process
- **HSP**: haemodialysis if indicated by severity of acute kidney injury ◈
- **INR** > 20 without bleeding or > 2 with severe bleeding: FFP 10–20 ml/kg ◇, vitamin K 5–10 mg IV (stop warfarin) ◇
- **Heparin overdose**: protamine 1 mg/100 units heparin ◈
- **Haemophilia A**: if bleeding or at risk for severe bleeding (e.g. blunt head injury in known haemophiliac), factor VIII replacement ◈. Many products available; vary per hospital. If no product, use high-dose FFP; monitor for volume overload
- **Haemophilia B**: if bleeding or at risk for severe bleeding, recombinant factor IX ◈. If unavailable, prothrombin complex concentrate (PCC) ◈. Desmopressin (dDAVP) may be used for bleeding in mild disease ◈
- **von Willebrand's Disease**: desmopressin 0.3 mcg/kg (max 20mcg) IV infusion, repeated every 12 hours as needed
- **Severe bleeding**: tranexamic acid 25 mg/kg every 6–8 hours oral or intravenous

DIC

The goal of acute management is haemostasis, correction of coagulopathy, and treatment of the underlying condition.
- VS, haemostasis, correct hypovolaemia. Treat the cause
- If bleeding predominates, platelets (if platelets < 50 000), FFP, fibrinogen concentrate/cryoprecipitate, vitamin K 5 mg IV, /folate
- If thrombosis predominates, consider therapeutic IV heparin 500 U/hour (no initial bolus)

Critical documentation

Severity of current bleeding; blood products used, lab results.

Disposition

Admit (to Haematology if available) if critical anaemia or thrombocytopaenia, appropriate therapy not available, toxic appearance, or severe bleeding.

118 Emergency use of blood products

Types of blood product

	Whole blood	Packed red blood cells	Platelets	Fresh frozen plasma (FFP)	Cryoprecipitate
Volume per unit	450 ml Hct 0.35–0.45	240–340 ml Hct 0.65–0.75	25–50 ml	250 ml	15–25 ml
Use	Needed urgently and/or individual elements not available	Acute or chronic anaemia	Prevent and treat bleeding due to thrombocytopaenia or abnormal platelet function	Treat coagulopathies (DIC, liver disease, factor deficiency, warfarin excess); Haemophilia B (if no factor IX concentrate)	Contains fibrinogen, von Willebrand factor, factor VIII. Used for DIC, haemophilia A (if no factor VIII concentrate), von Willebrand's (if no DDAVP)
Blood typing required	Yes	Yes	Preferred	Yes	Yes
Cross-matching required	Yes	Yes	No	No	No
Rh-matching required	Yes	Yes	Yes	No	No
Adult dose	2–4 units	4 ml/kg (1 pack) over 1–4 hours Each pack raises Hgb by ~1 g/dL	6–10 units over 1 hour Each unit increases platelets by ~5 000	10 ml/kg over 1 hour 2.5% factor increase per 1 unit	6–12 bags at rate 6 bags per hour 350 mg fibrinogen per unit
Paediatric dose	20 ml/kg	10 ml/kg over 2–4 hours	1 unit/10 kg over 1 hour	10 to 15 ml/kg over 1 hour	1 unit/10 kg over 1 hour

(hct = haematocrit)

Coagulopathy correction

- Factor VIII concentrate: haemophilia A; use per local protocol
- Factor IX concentrate: haemophilia B; use per local protocol
- Recombinant Factor VII: haemophilia with factor antibodies, Factor VII deficiency; use per local protocol
- Complications: thromboemboli, development of factor antibodies, allergic reaction

Before transfusing

Blood grouping and cross-matching ('type and cross')

Matches ABO blood group, Rhesus D type, RBC antibodies. Incorrect grouping results in life-threatening RBC haemolysis.

Cross-matching identifies possible transfusion reaction between donor and recipient blood.

- Rh+ blood may be transfused to Rh- recipients if Rh– blood supply is limited. Risk is in haemolytic reaction with **future** transfusions. **In event of emergency, this risk may be justified**
- If Rh– product is given to Rh+ child or woman < 50 years, Rho(D) immune globulin must be given to minimise future haemolysis
- If Rh– product given to Rh+ male or female > 50 years, Rho(D) immune globulin may be given

Special preparation

Leukoreduction removes white blood cells. Minimises sensitisation in patients receiving large numbers of transfusions (e.g. sickle cell, leukaemia).

Irradiation limits graft-versus-host disease. Used for patients who might receive bone marrow transplantation.

Complications of blood products

Volume overload

Children and elderly particularly vulnerable. Avoid giving large volumes **rapidly**, monitor closely (crackles, tachypnoea, tachycardia, hypoxia).

- Furosemide 2 mg/kg IV and pause infusion (see CCF, 🕮 p. 104)

Hypothermia

Large volume infusion of chilled blood. Infants at particularly high risk. **Use blood warmer**.

Citrate toxicity

Citrate preservative in blood binds calcium and magnesium, resulting in hypocalcaemia and hypomagnesaemia in patients receiving large volumes of product.

Hyperkalaemia

Stored red blood cells leak potassium, with older (> 7 days) blood higher risk.

Blood-borne infections

Potentially transmitted infections include HIV, hepatitis B and C, syphilis, malaria, HTLV, trypanosomiasis, and CMV. Reduced significantly by donor screening and testing. Bacterial contamination is limited by proper preservation and refrigeration.

Transfusion reaction

3% of individuals receiving blood products will have an adverse event. Those who have received previous transfusions are at highest risk.

The first five minutes

- **Stop transfusion immediately** if fever, chills, hypotension, pain, respiratory distress, bleeding/oozing, hives
- ABC, VS, O_2, cardiac monitor
- Inform lab, repeat cross match; blood cultures, document reaction

	Cause	Syndrome	Time to onset	Signs and symptoms	Management
Haemolytic reaction	Acute: ABO incompatability Delayed: RBC antigen-antibody response Non-immune: hypotonic fluids, temperature change, or mechanical shearing of blood cells during transfusion	Haemolysis If acute, DIC	Acute: minutes Delayed: days to months Non-immune: hours	Falling Hgb Jaundice Indirect hyperbilirubinemia Elevated LDH Acute kidney injury Haemoglobinuria Acute and delayed types may have fever	Stop transfusion Alert blood bank Supportive care IVF (isotonic) Monitor urine output Supportive care for ensuing DIC Aggressive IVF to urine output 3-5 ml/kg/hour for haemoglobinuria Furosemide if volume overload ◇ Identify antibodies ◈ Prevention: good transfusion technique Type and cross-match all blood Use large-bore IVs, no kinks or twists in lines Avoid simultaneous hypotonic fluids
Anaphylaxis (📖 p. 38)	Antigen-antibody reaction	Anaphylaxis.	Seconds to minutes	Shock Hypotension Tachycardia Angio-oedema Respiratory distress Cardiac arrest	Life-threatening. Stop transfusion High-flow O_2 Adrenaline 0.3 ml 1:1 000 IM (adults) IVF bolus Hydrocortisone 5 mg/kg, max 250 mg Alert blood bank - subsequent blood product should be washed.
Transfusion-related acute lung injury (TRALI)	Unknown Likely donor antibodies or cytokine reaction	Noncardiogenic pulmonary oedema	Hours	Respiratory distress CXR: bilateral fluffy infiltrates suggestive of oedema	Supportive care High-flow O_2 Typically resolves 24–48 hours Respiratory support as required
Febrile reaction	Mediated by cytokines	Most common reaction Occurs in up to 30% of platelet transfusions	Minutes	Fever	Antipyretics Hold transfusion for one hour If haemodynamically stable with fever resolved and no other symptoms, resume transfusion at slower rate, monitoring carefully. If repeat reaction, discontinue transfusion.
Urticarial reaction	Triggered by donor plasma proteins	Histamine release	Minutes	Hives Itching Laryngeal oedema Bronchospasm	Stop transfusion Diphenhydramine ◇ Hold transfusion one hour If haemodynamically stable with urticaria resolved and no dyspnoea, hypotension, or anaphylaxis, resume transfusion at slower rate. If repeat reaction, discontinue transfusion.

Critical documentation

Signs and symptoms, reaction type, serial VS and interventions. Fill request forms for investigation of adverse event, inform lab.

Disposition

Admit following severe reaction.

119 Clinical approach to breast lesions

History and physical exam

Key historical features

- Mass: time present, relationship to menses, skin changes, pain, nipple discharge
- Age, breastfeeding, menopause
- Family history breast, ovarian, colon cancer
- Personal history of breast cancer, masses/biopsy, recent breast trauma/surgery, recent pregnancy, abnormal mammograms
- Current use of hormone therapy or oral contraceptive pills

Signs and symptoms

- Evaluate breast symmetry with patient sitting and lying
- Palpate nipple/areola for discharge
- Evaluate lymph nodes of the axilla, supraclavicular area, neck, chest wall
- Note mass characteristics: mobile vs. fixed, firm vs. rubbery, oedema, inflammation, skin pitting, ulceration

Possible causes and differential diagnosis

Infectious and inflammatory

Erythematous, tender, warm.

Mastitis

Characteristics: painful, red, inflamed breast tissue. Common during **breastfeeding**. Associated **malaise, fever, chills, headache**.

- Investigations: CBC (infection, if ill-appearing)
- Management
  - » Majority of infections caused by *Staphylococcus aureus*
    - ▪ Antibiotics safe for breastfeeding – first-line antibiotics: cloxacillin, flucloxacillin. If suspect MRSA – clindamycin
    - ▪ If infant over two months – TMP/SMX
  - » Continue breastfeeding. Manually express milk if infant unable to suck
  - » Warm compresses, massage affected area, paracetamol

Breast abscess

Characteristics: painful, red, inflamed breast tissue with **fluctuant mass**. May drain pus; may be associated with severe mastitis.

- Investigations: CBC (if systemic symptoms)
- Management:
  - » Antibiotics

- Breastfeeding – see above. Not breastfeeding – MRSA: clindamycin, TMP/SMX, doxycycline; Gram negatives: fluoroquinolone or third-generation cephalosporin
- < 5 cm – needle aspiration; > 5 cm – I&D

Benign and malignant lesions

- Benign: smooth, soft to firm, mobile, well-defined margins, no skin changes
- Malignant: hard, immobile, fixed to surrounding skin and soft tissue, poorly defined or irregular margins, overlying skin changes, lymphadenopathy, nipple discharge

Differential diagnosis

Benign:
- Breast cyst: fluid-filled cavities varying in size with menses
- Fibroadenoma: firm, mobile mass in patients < 30 years old
- Cystosarcoma phyllodes: variant of fibroadenoma in patients > 30 years
- Fibrocystic changes: breast swelling or pain with menses
- Fat necrosis: firm, irregular mass with calcifications. History of local trauma
- Gynaecomastia: female breast tissue in men, due to oestrogen tumours, liver failure, decreased testosterone

Malignant:
- Infiltrating ductal: 80% of cancers, discrete mass
- Infiltrating lobular: 10%, often bilateral, thickening of breast, often no palpable mass
- Inflammatory carcinoma: 3%, most lethal, vascular and lymphatic invasion common, peau de orange (skin thickening), nipple retraction
- Paget’s disease: 2%, tender itchy nipple, occasional bloody discharge

Investigations

Mammogram ⟐ or breast US with guided-biopsy ⟐. Should **not** delay management.

Management

- Fibroadenoma, breast cyst, fibrocystic change: reassurance
- Fibrocystic changes: NSAIDs or oral contraceptive pills
- Fat necrosis, cystosarcoma phyllodes: elective excision of lesion
- Gynaecomastia: evaluation of underlying condition
- Suspected malignancy: rapid referral to surgery/oncology

Critical documentation

History, physical features of mass (so that evolution or change may be appreciated).

Disposition

Discharge and follow up if suspected infection or inflammation, suspected benign lesion. Admit if progression of symptoms, systemic illness, suspected malignancy (unless rapid out-patient follow-up available).

120 Oncologic and chemotherapy emergencies

The first five minutes

ABC, VS, ECG, IV access, IVF, cardiac monitor.

Oncologic emergencies

Febrile neutropaenia

Neutropaenia predisposes to severe septicaemia.

History and physical examination

Careful examination for infection source: pneumonia, spontaneous bacteraemia, perirectal abscess, typhlitis, intra-abdominal infection, UTI.

Investigations

As directed by suspected aetiology.

Management

The goal of acute management is supportive care and antibiotics until neutropaenia resolves. Use neutropaenic precautions.

- Broad-spectrum antibiotics covering gram positive organisms, *Staphylococcus*, gram negatives, anaerobes, and *Pseudomonas* (see Fever in immunodeficiency (🕮 p. 310)

Tumour lysis syndrome

- Massive cell death causes severe electrolyte derangements (hyperkalaemia, hyperphosphataemia, hypocalacaemia), hyperuricaemia, and acute kidney injury
- Classically occurs in bulky tumours (Burkitt's lymphoma) and leukaemia/lymphoma. Highest risk with first round of chemotherapy

History and physical examination

Often asymptomatic. Maintain high degree of suspicion.

Investigations

- Labs: electrolytes, renal ◇; phosphorus, uric acid, LDH ◈
- ECG: ◇ electrolyte-associated arrhythmias or changes

Management

The goal of acute management is correction of life-threatening electrolyte abnormalities and prevention of kidney damage.

Mainstay of treatment is prevention:

- Aggressive pre-hydration: 2–3 l/m^2 with goal urine output 2 ml/kg/hour
- Loop diuretic used only if required by volume overload
- Rasburicase and/or allopurinol in high-risk individuals ◈

Treatment of established tumour lysis syndrome:

- Aggressive hydration
- Aggressive management of hyperkalaemia (🕮 p. 426)
- Treatment of symptomatic hypocalcaemia (🕮 p. 423)
- Rasburicase 0.2 mg/kg IV daily × 5 days ◈
- If unable to obtain rasburicase and if patient is acidaemic, urinary alkalinisation with IV bicarbonate (goal urine pH 7.0)
- Haemodialysis if hyperkalaemia refractory to medical treatment, severely symptomatic hypocalcaemia, acute kidney injury ◈

Neutropaenic necrotising enterocolitis (typhlitis)

High mortality. Severe infection and inflammation of terminal ileum, caecum and appendix in neutropaenic patients, most often undergoing chemotherapy for leukaemia.

History and physical examination

- Fever, abdominal pain, vomiting, diarrhoea
- Abdominal tenderness, greatest in right lower quadrant; septicaemia or shock; peritoneal signs

Investigations

- Labs: CBC, electrolytes, blood cultures ◇
- Imaging: contrast CT abdomen/pelvis ◈

Management

The goal of acute management is treating acute intra-abdominal infection and associated septicaemia.

- Supportive care: IVF, analgesia
- Broad-spectrum antibiotics to cover gut flora (gram negative and anaerobic)
- Serial abdominal examinations for development of peritonitis
- Surgical consultation. Indications for operative intervention include intra-abdominal haemorrhage, bowel perforation, or infection not responsive to full medical management

Hypercalcaemia

Most common electrolyte abnormality in malignancy. Acute onset.

- Causes:
  - Osteolytic activity: multiple myeloma, lymphoma, bony metastases
  - Paraneoplastic syndromes:
    - Vitamin D-secreting lymphoma
    - Pseudo-hyperparathyroidism (PTHrP): renal, ovarian, breast, uterine, cervical, esophageal, lung, head and neck cancers
  - Tumour lysis syndrome

See Hypercalcaemia (📖 p. 423) for details.

Superior vena cava syndrome

Compression of superior vena cava by obstructing mass (most common) or vascular endothelial invasion. Most commonly lung, breast, mediastinal tumours (e.g. lymphoma). Differential includes TB or syphilitic aneurysm.

History and physical examination

- Face, head and neck, upper extremity swelling
- Dyspnoea, cough, headache
- Hypotension due to decreased right heart filling
- Symptoms exacerbated by leaning forward or lying flat. Improved with sitting up, standing up
- May be associated with right pleural effusion or oesophageal varices

Investigations

Imaging: contrast CT neck and chest ◈

Management

The goal of acute management is symptom relief and maintaining circulation.

Minimise oedema:

- Elevate head
- Consider high-dose steroids and loop diuretic
- Consultation to oncology ◈
- Consultation to interventional radiology or vascular surgery ◈
- Appropriate treatment per specific tumour type

Hyperviscosity syndrome

Increased blood viscosity due to cancer-related proteins results in sludging, poor perfusion.

Symptoms related to small blood vessel hypoperfusion: CNS, cardiovascular, visual. Most common in Waldenstrom's macroglobulinemia, multiple myeloma. Cause of death usually intracranial haemorrhage or respiratory arrest.

History and physical examination

Visual changes, headache, confusion, coma, dyspnoea, chest pain, congestive heart failure, acute kidney injury, ischaemic stroke, myocardial infarction.

Investigations

- Labs: CBC, electrolytes, renal ◇; PT/PTT ◈
- Imaging: CXR ◇ (if chest pain or dyspnoea); CT head (if AMS) ◈
- ECG

Management

The goal of acute therapy is maintaining cerebral and cardiac perfusion without causing volume overload, while treating hyperviscosity. Initiate treatment for underlying malignancy.

- Temporising measures while awaiting transfer: aggressive hydration, consider phlebotomy
- Plasmapheresis ◈

Hyperleukocytosis syndrome

Similar to hyperviscosity syndrome but differs in aetiology: leukostasis due to severely high white blood cell count of leukaemia rather than paraproteinaemia.

Most common in myeloid-type leukaemia (AML, CML). May be associated with tumour lysis syndrome or DIC.

History and physical examination and Investigations

See 'Hyperviscosity'.

Management

In addition to 'Hyperviscosity':

- Emergency chemotherapy ◈
- Leukopheresis ◈
- Hydroxyurea ◈

Epidural spinal cord compression

Tumour mass compresses spinal cord, resulting in rapidly progressive and severe neurologic sequelae. Often due to bulky lymphoma or spinal metastases from prostate, breast, or lung cancer. See Spinal cord compression (📖 p. 460 for details).

Cardiac tamponade

(📖 p. 102)

Disseminated intravascular coagulation

Most often associated with acute leukaemia. (📖 p. 288)

Complications of chemotherapy

Thrombocytopaenia

(🕮 p. 288)

Low platelets predispose to severe bleeding, including spontaneous intracranial bleed and massive haemorrhage with minor trauma.

History and physical examination

Gum oozing, gastrointestinal bleeding, epistaxis, AMS, any trauma or injury, excessive bruising.

Investigation

• Labs: CBC with peripheral smear, group and type ◇, PT/PTT ◈

Management

The goal of acute management is prevention of bleeding. Use full precautions

• Platelet transfusion ◈:
  » for < 10 000 in absence of bleeding
  » for < 50 000 if bleeding or minor procedures
  » for < 100 000 if severe bleeding

Anaemia

(🕮 p. 285 and 🕮 p. 292)

Mucositis

History and physical examination

Breakdown and ulceration of mucous membranes. Painful.
Oral mucosa, intestinal lining, rectal mucosa may be involved.

Management

The goal of acute management is pain control while limiting extent of membrane destruction.

• Hydration
• Oral care with salt and baking soda or chlorhexidine to prevent infection
• Stool softeners
• 'Magic mouthwash' for oral pain: many formulations available:
  » Often includes antacid, topical antibiotic and antifungal, topical steroid, viscous lidocaine for analgesia

Nausea and vomiting

History and physical examination

Dehydration, AMS, weakness suggestive of electrolyte abnormalities, abdominal pain, other complications cause of weakness/nausea/vomiting (e.g. infection).

Investigations

• Labs: electrolytes as indicated ◇
• Infectious work-up if suspected

Management

The goal of acute management is palliating nausea/vomiting, treating dehydration and electrolyte abnormalities, and encouraging adequate nutrition.

• Anti-emetics:

» Metoclopramide or promethazine or prochlorperazine
» If no relief, add benzodiazepine or diphenhydramine
» If no relief, ondansetron ◇

- Hydration, electrolyte replacement
- Pre-chemotherapy prophylaxis: high-dose IV ondansetron ◈

Critical documentation

Type of malignancy, diagnosis and treatment history, current chemotherapy regimen, date of last chemotherapy, history of complications. Investigation results, treatments and response, urine output.

Disposition

Admit all patients to the level of care with resources necessary for treatment. Consult with patient's treating oncologist where possible.

121 Paediatric oncology presentations

Including malignancy in the differential diagnosis of a sick child can lead to earlier diagnosis and referral, and may improve outcomes. (📖 p. 298.)

The first five minutes

- ABC, VS, IV, cardiac monitor ◇
- Evaluate and treat for other causes of the child's presentation (such as infection)

Key historical features

- Fatigue, pallor, bruising, fevers, night sweats, malaise, weight loss
- Duration of symptoms
- Development of masses, increase in abdominal girth, lymphadenopathy
- Unexplained limp, or focal bone pain

Acute leukaemia

Caused by uncontrolled growth of immature white blood cells in bone marrow, which suppresses normal haematopoiesis. It is the most common paediatric malignancy worldwide.

Signs and symptoms

- Pallor, fatigue, fever, bruising, petechiae, purpura
- Extramedullary involvement – hepatomegaly, splenomegaly and LAN
- Bone pain – leukaemic involvement of the periosteum

Investigation and treatment

- Labs: CBC (with differential and smear; blast cells (immature forms) in peripheral blood; anaemia and thrombocytopenia (bone marrow replacement by abnormal cells)), renal, type and cross ◇; LFT, PT/PTT, bone marrow testing ◈
- > 90% can experience complete remission with early treatment

Hodgkin's disease and non-Hodgkin's lymphoma

A malignancy of lymph nodes, spreads to other nodes and lymphatic channels. Reed-Sternberg is the malignant cell.

- Non-Hodgkin's lymphomas – heterogeneous group of malignancies of lymphatic tissue. HIV and EBV are associated
- Burkitt's Lymphoma is the most common paediatric malignancy

Signs and symptoms

- Supraclavicular or cervical LAN (often painless)
- Splenomegaly (more advanced disease)
- Systemic symptoms (fever, fatigue, night sweats, weight loss)

Investigation and treatment

Definitive diagnosis is based on biopsies done in specialty centres.

- Labs: CBC (anaemia and thrombocytopenia) ◇
- Imaging: CXR ◇; CT ◈ (both show masses)

Central nervous system (CNS) tumours

Second most common group of paediatric malignancies.

- Supratentorial: cerebral astrocytomas, optic gliomas, and craniopharyngiomas
- Infratentorial: cerebellar astrocytoma, brainstem glioma, ependymoma, medulloblastoma

Signs and symptoms

- Headache, vomiting, anorexia, irritability
- Seizures – focal or generalised
- Vision changes
- Ataxia or incoordination

Investigation and treatment

- Imaging: CT or MRI ◈
- Elevated ICP should be treated and emergency neurosurgery consultation obtained
- Seizure treatment (📖 p. 76).

Wilm’s tumour (nephroblastoma)

Most common paediatric abdominal malignancy; arise from embryonic renal cells.

Signs and symptoms

- Non-tender or tender abdominal mass
- Abdominal mass typically does not cross the midline

Investigation and treatment

- Imaging: US ◇; CT or MRI (all are suggestive)

Neuroblastoma

Malignant tumour arising from sympathetic neuroblasts/embryonic renal cells in the adrenal medulla and sympathetic chain. Two thirds arise in the pelvis and abdomen.

Signs and symptoms

- Abdominal mass that may cross the midline
- Skin manifestations can include bluish, non-tender subcutaneous masses
- Mass effect can cause renal and hepatic complications

Investigation and treatment

- Imaging: US ◇; CT or MRI ◈

Primary bone tumours

Ewing’s sarcoma and osteosarcoma common: osteosarcomas – metaphysis; Ewing’s can also arise in extraosseous tissues.

Signs and symptoms

- Local pain at site of tumour
- Limp or change in use of a limb
- Mass, pathologic fracture, tenderness
- Fever

Investigation and treatment

- Imaging: XR (suggestive) ◇; CT or MRI (definitive) ⟐

Rhabdomyosarcoma

Malignant solid tumour that often arises from the mesenchymal tissue that normally forms striated muscle.

Signs and symptoms

- Most commonly present as painless masses in head and neck
- Masses of the extremities and trunk are also possible
- Metastasis to bone, lungs, lymph nodes, bones, bone marrow, brain, spinal cord and heart can cause focal symptoms

Investigation and treatment

- Imaging: CT or MRI (typically diagnostic) ⟐

Retinoblastoma

- Most common intraocular tumour of childhood
- Arising from abnormal cells in the retina

Signs and symptoms

- Leukocoria or strabismus are the most common presenting signs
- Vitreous haemorrhage, orbital cellulitis, and microphthalmos are also possible presentations

Investigation and treatment

- Imaging: CT or MRI (determine presence of choroidal, orbital, subarachnoid, intracranial, or optic nerve involvement) ⟐
- Immediate referral

Critical documentation

- Clinical findings, imaging results, laboratory results
- Differential diagnosis, including oncologic possibilities
- Presence or absence of signs of metastatic disease or oncologic complications

Disposition

Early treatment of paediatric malignancies leads to better outcomes. Admit all patients with suspected malignancy to paediatric oncology where available.

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Part

4

H. Infectious diseases

122 Fever in the patient with immunodeficiency

Single temperature > 38.3 °C or a sustained temperature > 38.0 °C for > 1 hour in a patient with absolute neutropaenia (ANC < 500) or HIV with CD4 < 200. Patients are at high risk for bacterial sepsis with rapid decompensation.

The first five minutes

- ABC, VS, facemask O_2, IVF for shock, monitor
- Rapid risk assessment (immunodeficiency type, fever duration, antimicrobial prophylaxis, indwelling catheters, exposures)
- Initiate empiric broad spectrum IV antibiotics

History and physical examination

Infections may present atypically in the immunocompromised.

Key historical features

- Duration of fever; recent instrumentation or surgical procedures
- Current medications, including ARV (note most recent CD4 and nadir) or prophylactic agents, chemotherapy regimen (note most recent treatment, anticipated nadir)
- History of previous neutropenic fever or opportunistic infections with specific agents (fungal, etc.) if known

Signs and symptoms

- General appearance (non-toxic, toxic). Mental status, headache, neck pain
- Oropharynx, sinuses, dentition, oropharyngeal pain or ulcers
- Cardiac murmurs, septic embolic phenomena (splinter haemorrhages, janeway lesions, osler nodes, conjunctival haemorrhages)
- Crepitations, hypoxia, respiratory distress, cough
- Abdominal tenderness or distension, bowel sounds (severe immunosuppression may mask peritonitis), vomiting, diarrhoea, constipation
- Dysuria, vaginal discharge, genital cellulitis or pain
- Full skin exam for possible abscess (including perirectal) or necrotising fasciitis. Never perform a rectal exam in a neutropaenic patient

Possible causes and differential diagnosis

- Guided by signs and symptoms, but remember that focal findings may not present until immune reconstitution
- Always consider bacteraemia, pneumonia, UTI, CNS infection, abdominal catastrophe, sinusitis, severe cellulitis, endocarditis, abscess (dental, abdominal, perirectal, scalp)

Investigations

All febrile patients with immunodeficiency should get a basic workup, regardless of symptoms: CBC w/differential, chemistry, renal function, LFTs, malaria, UA, CXR ◇. Other investigations guided by history and physical:

- HIV+: CD4, viral load, sputum for TB/PCP ◇; serum cryptococcal antigen ◈
- CSF, including cell count, AFB, India ink or CrAg, stains/cultures ◇
- Bacterial, viral, fungal stains/cultures, including AFB: blood, urine, sputum, stool, wounds
- Imaging:
  - » US abdomen: cholecystits, perinephric abscess ◇
- Advanced imaging ◈:
  - » CT head/face: evaluate sinuses and brain for fungal or bacterial infection, toxoplasmosis
  - » CT chest: fungal disease, mycobacterial infection
  - » CT abdomen/pelvis: typhlitis, colitis, pancreatitis, abdominal abscess

Management

The goal of acute management is early identification and aggressive treatment of infectious, and screening for non-infectious causes of fever.

Remove any potential sources of infection (e.g. Foley catheter, cellulitic central line).

Antibiotics

Broad-spectrum empiric antibiotics until testing confirms source (📖 Antibiotic guidelines p. 968). If patient already taking prophylactic antibiotics, choose a different class for treatment. Chemotherapy treatment centres will usually have a neutropenic fever treatment protocol. If protocol unavailable, general guidelines include:

- Gram positive coverage: cloxacillin OR vancomycin (covers resistant pneumococcus and MRSA) for soft tissue infection, indwelling line or catheter, mucositis, pneumonia, ill appearance, or sepsis
- Gram negative and anaerobe coverage: (ceftriaxone AND ciprofloxacin) OR piperacillin-tazobactam
- Double gram negative coverage with anti-pseudomonal activity: (cefepime or meropenem) AND (gentamicin OR ciprofloxacin)
- Pen allergic: (ciprofloxacin AND clindamycin) OR (aztreonam AND vancomycin)
- Consider acyclovir and fluconazole if oral ulcers present
- Avoid chloramphenicol (bone marrow suppression)
- If no IV antibiotics available, ciprofloxacin AND amoxicillin-clavulanate with emergent referral

Antimalarials, antivirals, antifungal

- In endemic areas, test for malaria at presentation and treat empirically until repeat blood smear at 24h. Treat full course if testing unavailable.
- Consider adding antiviral therapy (e.g. acyclovir) for HIV patients with AMS.
- Consider adding empiric anti-fungal therapy if prior documented fungal infection or not improving on antibiotics

Consider non-infectious causes of fever

- Drugs/toxins, environmental, exposures, malignancy, hyperthyroidism

Critical documentation

Serial VS, cause and degree of immunocompromise, known exposures, home treatments, results pending investigations, treatments given and response.

Disposition

Admit all patients with fever and immunodeficiency. If signs of sepsis or shock, admit to ICU ⟐.

123 Approach to the adult with fever

Fever is defined as a core body temperature above 38°C, and is one of the most common acute presentations. See 📖 RAP for Adult fever p. 64.

The first five minutes

- ABCs, VS, O_2 for hypoxia, IVF for shock

History and physical examination

Key historical features

- Onset (sudden vs. gradual), duration (acute vs. prolonged (> 7–14 days)), pattern (persistent, intermittent, relapsing), travel history, sexual activity, medication history
- Co-morbid conditions: diabetes, HIV, chronic liver or renal disease, CCF

Physical examination

Thorough exam to localise source.

Systemic exam

- Eyes: conjunctival injection, jaundice, pallor
- Oral cavity: tonsils for erythema, oedema; look for thrush
- Ears: swollen and hyperaemic tympanic membrane and discharge
- Respiratory: rate, effort, and breath sounds
- Cardiovascular: pulse (for every degree rise in temperature, pulse rises by 10: dissociation is seen in typhoid, brucellosis, leptospirosis), examine cap refill, hypotension (signifies septic shock but is not always present), pericardial rub, murmurs
- Abdominal exam: organomegaly, tenderness, masses, ascites
- CNS: mental status, neck stiffness, focal neurologic deficits, pupil size and reactivity, fundoscopy
- Musculoskeletal: muscular tenderness, swelling, joint effusion and ROM, spinal tenderness
- Urogenital: urine amount (decreased is evidence of dehydration or shock with renal failure) and appearance (cloudy → infection, dark → dehydration or rhabdo); genital discharge or ulcers
- Skin: jaundice, rash, haemorrhage, cellulitis
- Nails: splinter haemorrhages (endocarditis)
- Lymph: diffuse LAN (cancer, TB, acute HIV)

Possible causes and differential diagnosis

See RAP for Adult fever p. 64.

Investigations

- Many diagnoses can be made accurately based on history and exam
- Every febrile patient in a malaria endemic area needs a thin and thick blood smear or Rapid Diagnostic Test (RDT)
- WBC may be high or low in infection and may be elevated by noninfectious conditions that stress the body (e.g. exercise, seizure), so often unhelpful
- Parasitic infections cause eosinophilia primarily when the parasite is migrating through body tissues. Patients with chronic parasitic infections often do not have eosinophilia

Associated signs and symptoms	Potential diagnoses	Investigations to consider	Notes
Headache	Meningitis (bacterial, viral, TB, fungal), malaria, viral syndrome, typhoid, dengue, leptospirosis, VHF	Blood slide, LP ◇ Blood culture ◇ CT scan ◈	Blood cultures have moderate sensitivity for typhoid CT head with and without IV contrast if HIV+
Sore throat, dysphagia, change in voice	Pharyngitis, epiglottitis, pharyngeal abscess	Soft tissue lateral neck XR ◇ CT scan ◈	XR with neck extended for assessment of pharyngeal soft tissues
Cough, sputum production, haemoptysis, SOB, chest pain	Pneumonia, TB, empyema, PCP	CXR ◇ Pleural fluid analysis ◇	HIV+ patients with TB do not have classic appearing XR; PTX suggests PCP
Chest pain, prolonged fever, multiple remote sites of infection	Endocarditis	Echocardiogram ◈ Blood cultures ◈ ESR ◇	Not all patients will have a murmur on exam
Right upper quadrant pain	Hepatitis, cholangitis, acute fasciola or other liver fluke, infected echinococcal cyst; liver abscess; visceral leishmaniasis	US ◇ Abdominal CT scan ◈ CBC and differential ◇	Eosinophilia may be absent in chronic parasitic infections; it is present when parasites are traversing tissues
Right lower quadrant pain	Appendicitis, enteric fever, psoas abscess	US ◇ Abdominal CT scan ◈	Patients with peritonitis should go to theatre without further testing; psoas abscess – hip may be flexed
Dysuria, flank/back pain	UTI, pyelonephritis	UA (LE and/or + nitrites indicate infection)	UA: + leucocyte esterase and/or nitrites; or micro > 5WBC/HPF or bacteria
Back pain	Malaria, brucellosis, Pott's disease, epidural abscess, pyelonephritis, dengue	Blood slide/RDT, brucellosis antibody titer, UA, blood cultures vertebral XR ◇ CT, MRI ◈	If Pott's disease or epidural abscess suspected thorough neuro exam; brucellosis testing has limited sensitivity
Focal swelling, erythema, and/or pain	Cellulitis, abscess, pyomyositis,	US ◇	Cellulitis is clinical diagnosis – consider US to evaluate for abscess
Swollen joint with limited ROM	Septic arthritis	Arthrocentesis with gram stain and culture ◇	Do not traverse potentially infected skin to access joint
Rash (Approach to rash, p. 144)	Chicken pox, HIV, enteric fever, Kaposis' sarcoma, drug rash	HIV test	Patients with acute HIV sero-conversion may have negative antibody tests
Diffuse lymphadenopathy	HIV, cancer	CBC, HIV test	Patients with acute HIV sero-conversion may have negative antibody tests

Possible causes of prolonged fever > 7 to 14 days

Symptom	Differential
Headache	Sinusitis, malaria, meningitis, enteric fever
Cough	Pneumonia, bronchitis, TB
Chest pain	Pleural effusion, empyema, pericarditis, pneumonia
Back pain	Pott's disease (TB of the spine), brucellosis
Diarrhoea	Enteric fever, colitis, drug induced
Abdominal pain	Hepatitis, liver abscess, enteric fever, pelvic inflammatory disease, visceral leishmanisis

Management

The goal of acute management is temperature control and treatment of the underlying cause.

Temperature control

- Evaporative cooling: exposure, tepid light spray with fanning for T > 40 °C. Do NOT use cold spray. Do NOT place ice packs (may cause shivering and increase temperature) or wet cloths directly to patient (will not remove

heat effectively)
- Pharmacological: paracetamol, diclofenac, ibuprofen or aspirin

Treatment of the underlying cause

Treat cause as indicated (may not need additional treatment if cause is a simple viral fever). See 🕮 RAP for Adult fever p. 64 for guidance to specific chapters.

Critical documentation

Serial VS, mental status, RBG; abnormal findings on exam; HIV status.

Disposition

Admit patients with evidence of poor perfusion, respiratory distress, hypoxia, AMS.

124 Approach to the child with non-localised fever

Acute fever: T > 38.0 °C for < 5 days.

Assessment of the febrile child is directed at identifying serious bacterial infection (SBI: bacteremia, meningitis, pneumonia, UTI) and other dangerous endemic causes of fever (e.g. malaria, typhoid, brucellosis, viral haemorrhagic fevers). Managing febrile children who present without localising signs and symptoms is a particular challenge, as aetiologies range from benign viral illnesses to life-threatening infections. This chapter describes a general approach based on severity of clinical presentation in children with no evident source; the empiric antimicrobial regimen described below may not be relevant for older, well-appearing children with clear signs of viral syndrome or other focal illness. See 🕮 RAP for Child with fever p. 62 for direction to specific chapters.

The first five minutes

- ABC, VS
- If toxic (see box) → O_2 by facemask, IV/IO, blood glucose, IVF for shock, initiate empiric antibiotics and antimalarials. If no glucometer, administer empiric glucose. If no IV/IO access, administer 2–5 ml 50% dextrose or sugar solution to buccal mucosa with caution for aspiration

History and physical examination

Key historical features

- Subjective parental assessment of fever is usually accurate and should trigger work-up even without documented fever
- Fever onset (sudden vs. gradual), duration (acute vs. prolonged > 7–14 days), pattern (persistent, intermittent, relapsing); travel history, immunocompromise in child or mother

Signs and symptoms

- Undress child completely for examination
- Evaluate for focal signs and symptoms: headache, ear or pharyngeal inflammation, neck pain or stiffness, flank or abdominal pain, diarrhoea, rash, dysuria or frequency, focal swelling or tenderness

Categorising children with fever

- **Toxic appearance**: abnormal level of consciousness or lethargic, AMS, DIB, poor muscle tone, weak or no cry, multiple abnormal VS
- **Well appearing but high risk**: no toxic features, but any of the following: < 3 months, incompletely immunised, malnourished, known or suspected HIV, other immunosuppression

- **Well appearing and lower risk**: no toxic or high risk features.

Management considerations

- Consider all who have been in an endemic area within the past year to have been exposed to malaria
- Consider children who have completed immunisations only within the past month to be incompletely immunised
- Children < 1 year with meningitis may have no localising signs

Symptomatic management

- Paracetamol 15 mg/kg Q4h orally or rectally
- Remove excess clothing
- Evaporative cooling: exposure, tepid light spray with fanning for T > 40 °C. Do NOT use cold spray. Do NOT place ice packs (may cause shivering and increase temperature) or wet cloths directly to patient (will not remove heat effectively)

Table 124.1 Diagnosis and management of child with fever and no source

Category	Diagnostics	Management
Toxic appearing	Malaria, CBC, UA, LP, CX	< 21 days: ampicillin 50 mg/kg IV Q6h AND gentamicin 5 mg/kg (0–7 d) or 7.5 mg/kg (7–21 d) IV once daily > 21 days: ceftriaxone 50 mg/kg IV BID Malaria endemic area: Empiric malaria treatment × 24 h for all Initial test negative: admit, repeat blood slide in 24h Testing unavailable: full course empiric treatment
Well-appearing but high risk by age (< 3 m) or above criteria	Malaria, CBC, UA, LP, CX	< 21 days: ampicillin 50 mg/kg IV Q6h AND gentamicin 5 mg/kg (0–7 d) or 7.5 mg/kg (7–21 d) IV once daily > 21 days: ceftriaxone 50 mg/kg IV/IM BID Malaria endemic area: Empiric malaria treatment × 24 h for all < 6 m Initial test negative: admit, repeat blood slide in 24h Testing unavailable: full course empiric treatment
Well-appearing, low-risk and age 3-12 m	Malaria, CBC, UA	Ceftriaxone 50 mg/kg IV/IM daily (esp. if CBC > 15) OR close inpatient observation Treat malaria per testing results
Well-appearing and low-risk and > 1 year	Malaria UA in all girls	Manage per focal clinical findings Treat malaria per testing results 24h follow-up

CX: blood, urine and spinal fluid cultures

Disposition

Admit all toxic-appearing and high-risk children, and those with difficulty returning for 24 hour re-evaluation.

125 Approach to sepsis

Definitions for resource limited settings were proposed in 2012:

- Sepsis: definite, or strongly suspected infection, plus ≥ 2 of four criteria – HR > 90, RR > 20, temp < 36 or > 38, malaise/apathy
- Severe sepsis: above + organ dysfunction, abnormal bleeding, or hypotension
- Septic shock: sepsis + arterial hypotension + inadequate tissue perfusion after adequate fluid resuscitation.

The first five minutes

- ABC, VS, facemask O_2, two large-bore IV; cardiac monitor and IVF for shock (2 L in adults or 20 ml/kg in children, unless severely malnourished (see 🕮 p. 394)
- Prepare broad spectrum antibiotics for administration within first hour

History and physical examination

Key historical features

- Any sick contacts or recent illness. Localising symptoms to identify source, including skin and oral cavity
- History of immunocompromise or other chronic illness, especially DM, CCF, alcoholism, HIV, malignancy, chemotherapy, steroid use

Signs and symptoms

- Hypotension, tachycardia, tachypnoea, fever, AMS
- Clinical signs of inadequate perfusion include delayed capillary refill, skin mottling, cool skin, weak peripheral pulses, AMS, poor urine output (< 0.5 ml/kg/hr adults or < 1 ml/kg/hr in children)
- Evidence of infectious source may include meningeal signs/CSF appearance, peritoneal signs, pulmonary findings, skin wounds, urine appearance, oral inflammation, sinus tenderness

Investigations

- All patients with exposure to endemic areas should be tested for malaria
- When available, cultures of blood, urine, and CSF if indicated ◇
- Imaging: CXR ◇ in all patients. Other imaging studies (i.e. abdominal CT scan ◈ based on results of H&P
- Consider serial serum lactate or bedside US of IVC to monitor resuscitation (US for hypertension) ◈

Management

The goal of acute management is to restore perfusion to tissues, identify and treat infectious source, and counter effects of systemic inflammatory response.

Supportive care

- Resuscitate with normal saline until tissue perfusion adequate. There is some evidence that targeting volume resuscitation to specific parameters (lactate clearance, venous O_2 extraction, US of IVC) can improve outcomes
- In severe anaemia (Hgb < 5 mg/dl) or dengue shock syndrome, consider colloids ◇
- Children: 20 ml/kg bolus and re-assess perfusion
  - » Caution in severely anaemic children
- VS at least Q20 minutes
- Monitor for signs of fluid overload
  - » Hypoxia or crepitations
  - » Hepatomegaly (children)
- Add vasopressor ◇ if perfusion not restored with volume
  - » Minimal data to support one pressor over another
  - » Ideally given via central venous catheter ◈
- Consider steroids ◇ for patients with hypotension not responsive to volume and pressors
  - » Hydrocortisone (200 mg IV adults) or equivalent. May consider in children (1–2 mg/kg IV) though evidence very poor
- Supplemental O_2 to a target of sat > 90% ◇
  - » Head of bed to 30–45° or place patient in lateral position
  - » For respiratory failure, use non-invasive ventilation first if available and appropriate ◈

Treatment of infection

- Do NOT delay antibiotic administration to perform tests in critically ill patients
- For source-unknown sepsis, antibiotic choice depends on local resistance patterns, drug availability and HIV rates
- Cover gram negative, positive, and consider anaerobes (for abdominal, odontogenic, or sinus source) See 🕮 p. 968 for antibiotic regimens
- Always consider need for anti-malaria coverage
- Consider empiric antiviral therapy (acyclovir or valacyclovir) ◇ in immunocompromised patients with possible meningo-encephalitis
- Drain or debride any accessible source of infection. Remove indwelling catheters or lines associated with infection

Critical documentation

Serial VS, efforts to identify and treat the source of infection; response to resuscitation on serial exams (e.g. volume status, perfusion); anti-microbials given; pending results (cultures).

Disposition

Admit all patients with suspected sepsis.

126 Approach to HIV

Patients with HIV frequently present acutely for complications of HIV itself or of anti-retroviral therapy, and those with stable disease may present for complaints that are unrelated to HIV, but require specialised care given the underlying infection. (See also 🕮 Patients with HIV infection in the acute care setting p. 870).

The first five minutes

ABC, VS, IVF for shock; blood glucose if AMS; assess gross neurologic function and whether severely malnourished.

History and physical examination

Risk behaviours for HIV infection and approximate year of onset. History of opportunistic infections, most recent CD4 cell counts and HIV viral load (RNA), medications.

See Table 126.1 for specific historical and physical features.

Critical documentation

- Confirmation of HIV diagnosis, CD4 count and nadir, viral load
- Current ARV regimen, compliance, evidence of treatment failure, or any complications
- History of any opportunistic infections (e.g. oral candidiasis, herpes zoster, Kaposi's sarcoma, etc.) or TB and treatment record

Disposition

- Admit patients with acute infections in the setting of HIV and depressed CD4 counts
- Well-appearing patients with well-treated HIV (CD4 count near normal) and reliable access to follow-up care can be managed as outpatient

Table 126.1 Major acute HIV related problems

Main Sx	Diagnosis	History	Physical	DDX	CD4+ count	Investigations	Management	Notes
Headache (HA), AMS	Cryptococcal Meningitis (CCM)	Encephalitis; HA (frontal), fever; AMS, vomiting blurred vision neck stiffness	AMS, diplopia (CN Palsy) Nuchal rigidity and photophobia may be absent	TB or bacterial Meningitis Toxoplasmosis HIV and other viral encephalitis CNS lymphoma	Typically < 100	LP ◇ – India ink for organisms; high opening pressure CSF or serum cryptococcal antigen test ◈ Gold Standard is CSF culture for ◈	See 🕮 Antibiotics p. 968	
	TB meningitis	AMS, photophobia, known or clinical signs of TB, gradual onset, fatigue	Low GCS, confusion	CCM, encephalitis, CNS lymphoma	Any	LP ◇ – zinc stain; may have high opening pressure	See 🕮 Antibiotics p. 968	More common in children 0–4 yrs See 🕮 TB p. 337
HA - focal neuro-deficit	Toxoplasmosis	Fever, AMS, and/or focal neurological deficits, ± seizures	Focal deficits (paresis, speech problems or sensory loss)	Primary CNS lymphoma Brain abscess PML Cryptococcoma Stroke	Usually < 100	CT or MRI ◈: single or multiple contrast-enhancing lesions (< 2 cm) Evidence of toxoplasmosis by +IgG, & PCR of CSF ◈ Evidence of response to therapy	Pyrethamine + sulfadiazine + leucovorin Alternative: Pyrethamine + leucovorin + clindamycin TMP-SMX	Confirm regimen with local ID specialist if possible
HA	CNS lymphoma	Seizures may be the 1st manifestation. Personality changes, AMS, HA and focal deficits	Confusion, lethargy, aphasia, memory loss Hemiparesis Seizures, CN palsy Headaches Fever rare	Cerebral toxoplasmosis, abscesses, glioblastoma and cerebral metastasis of solid tumors	CD4+<50 cells	Cranial CT or MRI ◈ EBV DNA ◈ ± surgical biopsy	ARV therapy Cranial radiotherapy Methotrexate in some research studies	
HA, fever	Bacterial meningitis	Acute onset HA	May be normal; stiff neck; confusion	Other meningitis (cryptococcal, viral); malaria; typhoid	Any	LP ◇ – CSF with ↑ WBC. Gram stain often normal CSF culture ◇	See 🕮 Antibiotics p. 968	Cover *Listeria* in all HIV+ patients
Fatigue	Anaemia	Gradual onset, vague symptoms	↓ alertness, conjunctival or palmar pallor	Malaria, depression, electrolyte imbalance – e.g. Ca	Any	CBC, blood slide or RDT for malaria ◇; peripheral film	Transfuse if Hb < 5, or <6 and symptomatic, or for shock, respiratory failure	May be due to HIV itself or BM suppression from AZT, TMP-SMX
Severe fatigue	Lactic acidosis	History of d4T, ddI, ddC, AZT among ARV, nausea, vomiting, fatigue, weakness, Risk factors: female, pregnancy, older, use of ribavirin	High BMI, proximal muscle weakness, signs of liver failure, neuropathy	Tissue hypoxia sepsis, other shock, liver or renal failure, carbon monoxide poisoning, intoxication, lymphoma	Any	Lactate levels ◈	Discontinue ARV for ≥4w (till lactate normal) Substitute TDF or ABC for d4T,ddI or ZDV Avoid all future use of d4T/ddI	Most common with d4T/ddI High mortality Admit all
Oral lesions	Oral thrush	Burning sensation, decreased taste, or asymptomatic;	Plaques of various sizes (1 mm to large) that can be removed by scraping	Oral hairy leukoplakia; condyloma acuminatum	< 400	Clinical Dx, removal with dry gauze leaves a red or bleeding surface	Fluconazole 200 mg x1, then 100 mg per day for 3 weeks	Near 100% recurrence
	Oral hairy leukoplakia	Asymptomatic Extent of lesions varies day to day	White or greyish-white plaque appears 'hairy'. Most common on inferolateral surface of the tongue	Thrush, secondary syphilis	< 250	Clinical Dx. Does not rub off	Usually clears if on acycolovir. Can apply podophylin 25% in tincture of benzoin	Caused by a virus; often recurs
Dysphagia	Candida oesophagitis	Odynophagia, feeling that food gets stuck Nausea, poor appetite	May or may not have oral lesions	CMV or herpes esophagitis	< 100	Empiric treatment should improve in 72 hours. Endoscopy if not ◈	Oral fluconazole 400 mg x 1. Then 100–400 mg/day x 14–21 days	Occurs in 10-20% of patients with HIV
	Herpes oesophagitis	Dysphagia, odynophagia	Oral ulcers (often), focal pain, infrequent fevers. Thrush in < 5% Upper GIB	*Candida*, CMV, pill esophagitis (doxy or, tetracycline, clindamycin, ascorbic acid, iron) or food-related oesophagitis, apthous ulcers, GERD	< 200	Endoscopy ◈ with biopsy for Dx: erythema and erosions, usually small, shallow. Microscopy and culture	Odynophagia may improve with antacids mixed with viscous lidocaine. IV acyclovir if severely immunocompromised. ARV therapy	ARV therapy has reduced incidence; *Candida* not more common. Rule out pill oesophagitis if CD4 > 200

Cough, SOB	TB	Fevers, night sweats, weight loss, cough, haemoptysis	See 🕮 TB p. 337				See 🕮 Antibiotics p. 968	Hold ARVs until patient on ant-TB x 2 weeks
	Bacterial pneumonia	Acute high fever, painful breathing, productive cough	T > 39.5, AMS, ↑HR, ↑RR	PCP, TB, Viral pneumonia	Any	Clinical, CXR ◇ US ◇	See 🕮 Antibiotics p. 968	
	PCP	Triad of SOB, fever and non-productive cough. Subacute onset	Fever, ↑HR, ↑RR,± crepitations	PE, TB, CMV, bacterial pneumonia	< 200	CXR with bilateral, symmetric interstitial infiltrates, PTX; sputum studies ◇; bronchoscopy ◈	See 🕮 Antibiotics p. 968	May need PPV and ICU
	Pulmonary Kaposi's sarcoma	DIB, fever, cough, haemoptysis, or chest pain. History of purplish papular lesions	Pleural effusion with chest pain, hypoxaemia, acute respiratory failure	Bacillary angiomatosis, TB, fungal PNA Other malignancies	200–500	Biopsy ◈ CD4 and viral load, CXR ◇; biopsy ◈ Exclude other causes with Sputum and micro, AFB ◇, bronchoscopy ◈	Start ARVs and chemotherapy if: rapid, lymphedema, persistent on ART, IRIS Pegylated liposomal doxorubicin	Note NNRTI and PI based combinations preferred
Diarrhoea (🕮 p. 242)	Acute < 2w Persistent 14d–4w Chronic > 4w	Duration of Sx frequency and quality of stool, (e.g. bloody, watery), weight loss. medications Travel and sexual history	Height Weight Orthostatic BP Fever Abdominal tenderness Monitor vital signs closely	Bloody diarrhoea: *Shigella, Salmonella, Entamoeba* Fever common: *Shigella, Salmonella* Fever uncommon: Cryptosporidium Irritable bowel	< 100 for chronic cryptosporidosis & microsporidosis	Ova & parasite exam ◇ + testing for *Giardia, Cryptosporidia, Cyclospora, Isospora,* microsporidia ◈ Endoscopy for malabsorption, colitis ◈	See 🕮 Antibiotics p. 968 Supportive care: PO or IVF as needed	Most HIV-associated diarrhoea will require extended outpatient workup
Rash (See drug eruptions)	Steven-Johnson Syndrome	Drug exposure up to 3 wks before, Hx of TMP-SMX, NVP or rifampicin or ethambutol use	Rash, mucus erosions, with systemic signs	Erythema multiforme, pemphigus, toxic shock, dress	Any	Clincial Dx CBC, LFTs, CXR to evaluate other causes ◇ skin biopsy ◈	See 🕮 Drug eruptions p. 165	TMP-SMX, nevirapine are the most common causes
	Secondary syphilis	History of painless genital lesion, other STIs	Rough, reddish brown rash; may be on palms/soles; grey-white lesions in warm areas	SJS; drug rash;	Any	Clinical Dx VDRL or RPR ◇ Confirm positive VDRLs with FTA-ABS ◈	See 🕮 Antibiotics p. 968	
	Kaposis' sarcoma	Purpuric lesions; may bleed easily	Red, purple, brown or black popular lesions	Pyogenic granuloma; bacillary angiomatosis	Any	Clinical Dx	ARVs as above	
Focal swelling	Pyomyositis	Swollen painful mass May be sub-acute	Firm, swollen area,	Abscess; tumour	Any	None; can do gram stain ◇	I and D followed by antibiotics (cloxacillin)	
	Scrofula (TB)	Painless mass, sub-acute-chronic, often in neck; weight loss, chills	Fever, neck mass with no overlying skin erythema; skin eventually adheres to the mass	Tumour, cervical adenitis, infected branchial cleft cyst; lymphoma	Any	Needle aspiration with AFB staining ◇	Anti-TB medications as per local guidelines	Attempts at excision can lead to fistulas
	Lymphadenopathy (LAN)	Diffuse LAN; early finding after infection	Multiple lymph nodes	Lymphoma	Any	None	None	Consider acute seroconversion

127 Malaria

Very common in Africa. May progress rapidly to fulminant disease, especially in children, elderly, immunosuppressed, and immune-naive adults (travellers).

The first five minutes

- ABC, O_2,IV as needed. IVF if shocked unless severe anaemia; transfuse for shock with severe anaemia. Cardiac monitor if high-output cardiac failure. Rapid blood glucose and IV dextrose if < 3.5. If no IV/IO access, 2–5 ml 50% dextrose to buccal mucosa (caution for aspiration)
- Elevate head of bed 30°. Evaluate for increased ICP and impending herniation (unequal pupils, posturing) – see below

History and physical examination

Key historical features

- Residence in or travel to a malarial area within past year; chemoprophylaxis? 'Out-of-season' transmission is common; malaria is transmitted in all seasons
- HIV, malnutrition, heart disease, pregnancy, immunocompromised patients more likely to develop severe illness

Signs and symptoms

Uncomplicated malaria: generally well-appearing, with parasitaemia <5% (cut-off depends on local endemicity), no evidence of organ dysfunction, and able to take PO. If signs/symptoms below, or patient is high-risk, pregnant, or

ill-appearing, approach as complicated malaria.

Markers of complicated malaria

Many complications of malaria infection (capillary leak syndromes, organ dysfunction, infarcts) are related to ‘cytoadherance’ of RBC to small vessels. Markers of complicated disease include:

- Clinical: lethargy, AMS, seizures, respiratory distress, shock, pulmonary oedema, abnormal bleeding, jaundice, haemoglobinuria, severe pallor
- Biochemical: acute kidney injury, acidosis, acute hepatitis, hypoglycaemia, hypoxia, lactic acidosis
- Haematological: high parasite burden; severe anaemia

Differential diagnosis

Influenza, viral hepatitis, meningitis, septicaemia, pneumonia, gastroenteritis, typhoid, tick fever, viral haemorrhagic fever, acute HIV.

Clinical syndromes associated with Plasmodium species

- *P. falciparum*: cerebral malaria, hypoglycaemia, renal/respiratory failure, severe anaemia, vascular collapse/shock
- *P. vivax*, *P. ovale*: anaemia, hypersplenism; relapse up to 5 years after acute infection
- *P. malariae*: chronic asymptomatic parasitaemia, nephrotic syndrome
- Mixed infections are common

Investigations

Laboratory

- Malaria Rapid Diagnostic Test (RDT) with confirmatory blood smears
- CBC, electrolytes, renal function, hepatic function, urinalysis ◇; VBG/ABG, lactate, PT/PTT ◈
- Evaluate for meningitis in all cases of suspected cerebral malaria: LP ◇ (p. 826)
- Cross type if severe pallor ◇

Microscopy

- Gold standard diagnostic test
- Identifies species; quantifies parasitaemia
- Negative smear does not exclude malaria: repeat smear until patient improves or alternate diagnosis confirmed

Malaria RDT

- Most detect only *P. falciparum*
- Sensitive, but performance depends on correct storage, use, interpretation
- May be negative in infections with low parasite burden

Management

The goal of acute management is treatment of severe anaemia, early anti-malarial therapy, and supportive care.

Supportive care

Fever (p. 62 and p. 64)

Seizures (📖 p. 74 and p. 76)

- No seizure prophylaxis – treat only active seizure
- Check RBG and correct hypoglycaemia (5 ml/kg dextrose 10%)

Hypoglycaemia

- Recurrent, frequently seen in severe malaria. Common quinine side-effect
- Check RBG Q4h in patients with AMS, receiving quinine, signs of malnutrition, and immediately if seizing. If unable to do frequent checks, consider empiric dextrose-containing fluid (e.g. D5% NS) or buccal dextrose administration (1 ml 50% dextrose Q3–5 min)

Volume status

- Inability to take PO, fever, dehydration, shock increase requirements
- Shock necessitates fluids but anaemia and multi-organ dysfunction (ARDS, renal failure, capillary leak, acute anaemia) confer high risk. Use cautious IVF boluses (20 ml/kg), monitor throughout and reassess after each bolus
- Follow volume status with exam (rising heart rate, respiratory rate, creps, periorbital oedema), urine output (goal > 0.5 ml/kc/hr), and bedside US (📖 p. 798)

Severe anaemia

- Haemolysis may cause acute, severe anaemia, resulting in high-output heart failure. Patients with Hgb < 5 require urgent blood transfusion (see transfusion chapter)
- When possible, hold IVF until transfused, given risk for high-output heart failure and pulmonary oedema. If severely shocked with prolonged time to transfusion, consider 10 ml/kg bolus with close monitoring to temporise while awaiting blood
- Transfuse slowly over 2–4 hours, monitoring O_2 sat and respiratory status. Pause transfusion, administer furosemide for signs pulmonary oedema (1 mg/kg IV paeds; 20–40 mg IV adults). Resume transfusion slowly when respiratory status improved

Elevated ICP and impending herniation

- In rare cases with rapidly progressive course, recent initiation of therapy, and recent onset of neuro deficits, mannitol may be used as a temporising measure to allow antimalarials to take effect. Mannitol is unlikely to be useful in patients with prolonged course, those on treatment for > 48h, or those who present with evidence of herniation of unknown duration
- Steroids and dextran have not been shown to improve outcomes

Antibiotics

- Malaria and bacteremia often co-exist; do full bacterial workup
- For septic patients, initiate broad-spectrum empiric antibiotics while awaiting lab and culture results (see antibiotics guidelines)

Uncomplicated *P. falciparum* infection

- First-line: Artemether-lumefantrine (Coartem®): 6 doses over 3 days (stat dose, dose 8 hours later, then BID for 2 days). Taken with food/milk. See below for dosing
- Second-line: oral quinine 10 mg/kg TID × 7 days PLUS (7 days of either doxycycline 100 mg PO BID (contraindicated in pregnancy, children) or clindamycin 7 mg/kg (max 600 mg) PO TID × 7 days)

Dosage of artemether-lumefantrine (20 mg/120 mg)

- 5–15 kg: 1 tablet each dose; total 6 tablets
- 16–25 kg: 2 tablets each dose; total 12 tablets

• 26– 35 kg: 3 tablets each dose; total 18 tablets
• > 36 kg: 4 tablets each dose; total 24 tablets

Complicated *P. falciparum* infection

See text box complicated malaria.

IV Artesunate (preferred)

• Bolus injection: 2.4 mg/kg at 0, 12, 24 hours, then daily until taking oral. Then transition to full oral course

Quinine IV (alternate)

• Regional resistance
• Requires ECG, cardiac monitor ◇ for QT prolongation
• Frequent hypoglycaemia; monitor RBG
• Slow infusion. Load: 20 mg/kg in D5% over 4 hours
• Maintenance: 10 mg/kg IV every 8 hours until taking oral

Mixed infections/non-falciparum infections

• *P. ovale*, *P. vivax*: quinine, artemether-lumefantrine; then primaquine to prevent relapse
• *P. malariae*: chloroquine, quinine, artemether-lumefantrine

Prior treatment

In general, patients who have completed a course of therapy and present with persistent symptoms or have positive testing should be treated with a different category of drug. Partially treated patients who are improving should complete course. Partially treated patients who have persistent positive smear should be switched to a new agent.

Critical documentation

Means of diagnosis (RDT, smear) and results; treatments given (antimalarials, blood, adjunctive medications); volume status (IVF, urine output); comorbidities (HIV, heart disease, malnutrition).

Disposition

Discharge uncomplicated malaria after initial treatment dose if accompanied by reliable caretaker and means to return for worsening.

128 Meningitis

Meningitis is inflammation of meninges covering the brain and spinal cord, usually due to bacterial and viral infection, although may be caused by other pathogens and non-infectious agents.

Common causes of acute meningitis in adults and children

Bacterial	**Viral**
Streptococus pneumoniae	Enterovirus
Neisseria meningitides	Mumps, measles *, varicella, rubella *
Haemophilus influenzae	HIV (seroconversion illness)
Listeria monocytogenes §	Adenovirus *
E. coli #	Parainfluenza *
Group B *Streptococcus* #	Herpes virus

Mycobacterium tuberculosis	EBV *

\# In neonates

§ In neonates and elderly patients

* In children

The first five minutes

Isolate patients and use full droplet precautions for suspected bacterial meningitis. ABC, VS, IV, initiate IV antibiotics (📖 p. 968), IVF for shock.

Literature suggests that steroids may improve outcomes in *S. pneumonial* meningitis presenting early, but there are no Africa-based studies showing benefit. We do not recommend early empiric steroids. See 📖 Antibiotics p. 968 for recommendations in TB meningitis.

History and physical examination

Key historical features

Ask about recent URI, ill contacts, travel, time course of illness, immunocompromise, any neurosurgical procedures, baseline neurologic status.

Signs and symptoms

Onset within hours to days of two of the following symptoms occurs in 95%:
- Headache, fever, neck stiffness, AMS

Petechial rash (see purpura and rash chapters) is common in meningococcemia. Treat all patients with any of above signs and rash immediately with antibiotics.

Other common symptoms include photophobia, nausea and vomiting. Bacterial meningitis usually has a more fulminant presentation than viral or fungal – these may present sub-acutely with headache and AMS only. Neonates and young infants may present with non-specific features (poor feeding, irritability, inconsolable crying, lethargy, apnoea and seizures). In neonates and young infants, exam may be normal other than fever or hypothermia and irritability (classically worsen when being held). Bulging fontanelle is a late sign.

Possible causes and differential diagnosis

- Bacterial, viral, fungal, TB meningitis
- Drug-induced meningitis
- Carcinomatous or lymphomatous meningitis
- Secondary inflammatory meningitis (SLE, sarcoidosis, Beçhet’s disease, or Sjögren’s syndrome)
- Cerebral abscess
- Subarachnoid haemorrhage

Investigations

Diagnosis: LP with opening pressure and CSF analysis. See LP chapter 📖 p. 826 for procedure contraindications and CSF interpretation. Blood cultures may be useful if LP contraindicated.

Management

The goal of acute management is early aggressive antimicrobial therapy and supportive treatment to limit sequalae.
- Analgesia
- Antimicrobials: immediate empiric treatment. Delayed treatment is associated with poor prognosis. **DO NOT delay antibiotics for diagnostic testing.** See 📖Antibiotics p. 968 for agents and dosing
- For suspected bacterial meningitis, treat all exposed staff and patient household contacts with prophylaxis: ciprofloxacin 500 mg PO × 1 (adult) OR rifampicin (adults or children) 10 mg/kg (max 600 mg) PO BID × 2

days OR ceftriaxone 250 mg (≥ age 15) or 125 mg (< age 15) IM × 1.

Disposition

Admit all patients with suspected or confirmed meningitis and treat with antimicrobials.

129 Pneumonia in adults

Pneumonia is the leading infectious cause of death worldwide. Organisms vary widely based on regional context and vaccination patterns. Community-acquired pneumonia (CAP) in otherwise healthy patients is typically caused by *S. pneumoniae*, *Mycoplasma*, and viruses. TB (📖 p. 337) also is a common cause in Africa.

Mortality is increased in patients < 2, > 65, and with significant co-morbidities (e.g. malignancy, liver disease, renal disease, CCF, diabetes, alcoholism, sickle cell disease, other forms of immunocompromise). Poor prognostic signs include AMS, tachypnoea, hypoxia, hypotension, T< 35 or > 40, multilobar involvement, significant anaemia or uraemia (BUN > 30).

In patients with co-morbidities, *Haemophilus influenzae*, gram-negative bacteria, and *S. aureus* must be considered. In patients at risk for aspiration, also consider anaerobes and klebsiella. In current or recently hospitalised patients, consider *S. aureus*, anaerobes, *Pseudomonas*. *Legionella* pneumonia may occur in outbreaks. For patients with immunocompromise, see related chapters (📖 HIV p. 320, Fever in immunodeficiency p. 310).

The first five minutes

- ABC, VS, O_2 by facemask, IV. Look for respiratory distress (increased rate, hypoxia, accessory muscle use, retractions between the ribs or above the sternum/clavicles)
- Isolate those at risk for TB

History and physical examination

Key historical features

- Co-morbidities or immunosuppression, as above
- History of respiratory disease, including smoking, COPD, asthma, previous TB
- Elderly patients may present atypically with AMS or falls

Signs and symptoms

Assess general appearance, mental status, perfusion and hydration status.

- Symptoms: new cough or change in sputum (blood?), dyspnoea, rigors, sweats, fatigue, myalgias, anorexia
- Signs: fever or hypothermia, lethargy, diaphoresis, crepitations, dullness to percussion (effusion), increased tactile fremitus (consolidation)
- Respiratory distress: tachypnoea, accessory muscle use, hypoxia, cyanosis

Differential diagnosis

Bronchiolitis, asthma, COPD, CCF and pulmonary oedema, PE or infarction, PTX, SARS, influenza, malignancy, severe anaemia, MI.

Investigations

- CXR ◇ not necessary for diagnosis, but can identify extent, associated effusion or abscess, and alternate conditions (e.g. CCF, PTX). Obtain CXR in patients with immunocompromise or co-morbidities
- Concern for TB: sputum AFB stain × 3 ◇
- Respiratory distress or shock: CBC, chemistry, blood cultures ◇
- CAP is a common presentation of HIV; consider testing

Management

The goal of acute management is restoration of oxygenation and perfusion, and early treatment of infection.

General care

- O_2 for distress or low saturation ◇
- PPV for respiratory failure. Consider assisting 5–10 breaths hourly with bag valve mask (synchronise with breathing) to prevent air space collapse ◇
- NIPPV or intubation with mechanical ventilation for severe respiratory distress ◈
- Volume resuscitation for dehydration, sepsis, shock (See 🕮 Approach to sepsis p. 318) ◇

Antibiotics

See 🕮 Antibiotic guidelines p. 966.

Critical documentation

Serial VS, comorbidities, signs of respiratory distress, HIV status, TB exposure history.

Disposition

Admit for abnormal VS, DIB, hypoxia, bilateral pneumonia, immunocompromise, multiple comorbidities, high-risk features as above.

130 Pneumonia in children

Streptococcus pneumoniae, or pneumococcus is the most common bacterial cause of pneumonia, but viral causes are more common in young children. Mixed viral-bacterial infections are common in HIV-infected children.

The first five minutes

- ABC, VS, facemask O_2, IVF (20 ml/kg in less severely malnourished 🕮 152), cardiac monitor
- Administer antibiotics in ill-appearing children

History and physical

Key historical features

- Timing and duration of cough, DIB, fever, current or prior wheezing
- History of recurrent pneumonia or immunocompromise

Physical signs

- Tachypnoea (most sensitive and specific sign)
- Lower chest wall indrawing
- Other distress signs: grunting, nasal flare, use of auxiliary muscles, head nodding, central cyanosis, inability to feed, vomiting, poor capillary refill time, impaired consciousness

Differential diagnosis

- Severe pneumonia: lower chest wall indrawing
- Very severe pneumonia: central cyanosis, difficulty with oral intake, convulsions, lethargy, unconsciousness, or head nodding
- Bronchiolitis: wheeze and respiratory distress in infant < 2 years of age
- Asthma: recurrent wheeze, tight chest or cough; symptoms highly responsive to bronchodilator
- TB: cough ≥ 2 weeks; poor growth; TB contact

- Pertussis: mild illness course, cough ≥ 3 weeks, whooping cough
- Cardiac: cyanosis not correcting with O_2; tachycardia out of proportion to respiratory distress; history of chronic and progressive feeding difficulty, poor growth, breathlessness, hepatomegaly
- Metabolic acidosis: tachypnoea/hyperpnoea with normal work of breathing and normal lung exam
- Foreign body aspiration: wheezing, CXR findings

Investigations

- CXR ◇: always indicated for severe, very severe, or recurrent pneumonia; failed outpatient management; suspected PTB or foreign body aspiration
- HIV testing for severe, multi-focal, or recurrent pneumonia

Management

The goal of acute management is restoration of oxygenation and perfusion, and early treatment of infection.

All patients

- O_2: for sats < 92% on room air or signs of respiratory distress
- Intake: frequent small feeds; NGT if too distressed to feed. Reduce all maintenance fluids to 40–60% of normal requirements
- Paracetamol or ibuprofen for fever
- Antibiotics (📖 p. 966)

Additional treatment

- Symptomatic HIV infection, or severe malnutrition: add gentamicin for hospitalised children
- Suspected PJP: add cotrimoxazole (10 mg/kg IV load then 5 mg/kg/dose IV QD). Consider oral treatment (double IV dose) if not severe
- Suspected pertussis (whooping cough): add erythromycin 10 mg/kg/dose QD

Critical documentation

Severity assessment, medication doses, timings, response to treatment.

Disposition

- Admit: < 2 months; abnormal LoC; inability to feed or drink; requiring O_2 to maintain sats > 92%; severe pneumonia; severe malnutrition; failure to respond to outpatient care; clinical deterioration on treatment
- Admit to ICU (where available): sats < 90% on FiO2 > 70%; any apnoeic episodes; hypercarbia with acidaemia (pH < 7.25); progressive course with tiring
- Discharge: drinking well; sats > 92% on room air; tolerating oral medication
- Failure to improve after 48 hrs of treatment: review treatment; search for complications (para-pneumonic effusion, empyema, lung abscess, necrotising pneumonia); consider dual pathology or resistant organism

131 Tuberculosis

By the end of 2000, approximately eight million adults in Africa were co-infected with TB and HIV, with HIV conferring a ~50% lifetime chance of TB infection. TB presentations vary greatly, and immunosuppressed patients do not reliably manifest ‘classic’ TB symptoms. Maintain a high degree of suspicion for dual infection in HIV patients.

Classified by anatomical site (extrapulmonary or pulmonary); means of diagnosis (bacteriologic or clinical diagnosis); new or relapse; HIV status; and resistance patterns.

The first five minutes

- Isolate patient. Negative pressure room if possible. Evaluate patient under UV-light (direct sunlight) if provider N-95 mask not available
- ABC, VS, O_2 if hypoxic (caution for aerosolisation), IV. Evaluate for signs of respiratory distress or shock

History and physical examination

Key historical features

- High-risk groups: HIV+, prior TB, malnourished, smokers, diabetics, drug users, infants, elderly, and those in crowded living conditions (e.g. prisons, hostels, refugee camps) or with significant contact with TB+ patients (e.g. healthworkers)

Signs and symptoms

- Exam may range from normal to cachexia with severe respiratory distress
- Constitutional symptoms include fever, night sweats, fatigue, anorexia, weight loss
- Pulmonary TB: cough > 2 weeks, haemoptysis, chest pain, DIB. Exam may be unremarkable, or may have crepitations, wheezes, clubbing
- Extra-pulmonary TB: clinical syndrome varies by location, common presentations include abdominal pain and ascites; pericardial effusion; back pain ± weakness; subacute meningitis
- HIV+ TB: weight loss, fever predominate. Any HIV+ patient with cough, fever, weight loss/failure to thrive, or night sweats should be evaluated for TB
- Drug-resistant TB: must be considered in any patient with TB recurrence after treatment or with treatment failure (progressive symptoms or worsening CXR while on therapy)

Differential diagnosis

- Pulmonary: malignancy, including Kaposi's sarcoma; other pneumonia; interstitial lung disease; inhalational injury; bronchiectasis; reactive airways disease
- Extra-pulmonary: other causes of meningitis, pericardial effusion and tamponade, pleural effusion, ascites and peritonitis, hepatobilliary inflammatory syndromes, genitourinary infections, osteomyelitis (spine) and arthritis (hip, knee)

Investigations

- Pulmonary TB: sputum smears ◇ with culture for sensitivity testing ◈
- Extrapulmonary TB: fluid or tissue biopsy (i.e. CSF, bone, ascites, pleural or pericardial fluid) analysis ◇, with adenosine deaminase and culture and stain ◈
- Molecular testing (e.g. Xpert MTB/RIF ◇; nucleic acid amplification ◈)
- HIV testing for all TB+ patients
- Imaging: CXR (see box) ◇; CT chest ◈ more sensitive. Preferred when other co-existing lung disease or suspected miliary disease; CT imaging of suspected extrapulmonary sites (CNS, abdomen) ◈

XR findings in TB

Primary TB

- May be normal
- Parenchymal disease: favours middle and lower lobes; may appear as lobar/segmental atelectasis in children
- Hilar LAN: most common finding; unilateral > bilateral
- Pleural effusion: often unilateral; may be only sign of TB
- Miliary disease: diffusely scattered small nodules. More common in children, elderly and immunosuppressed patients. May represent primary or post-primary TB with spread

Post-primary TB

- Patchy consolidation primarily in upper lobes. May be in middle or lower lobes, particularly in HIV+ patients
- Cavitary lesions (less likely in HIV+ patients)
- Hilar lymphadenopathy ± RML collapse, solitary nodules, pleural effusion

Treatment

Management

The goal of acute management is to manage respiratory compromise, limit transmission, establish clinical diagnosis, initiate confirmatory testing and refer for therapy.

- Antipyretics (paracetamol), analgesia, and O_2 to maintain saturation > 90% ◇
- IV antibiotics for concomitant bacterial infections ◇. When TB suspected, avoid fluoroquinolones as empiric pneumonia treatment as they are important second-line agents for resistant TB strains
- Consider therapeutic thoracentesis (max 1.5 l) or paracentesis for significant dyspnoea ◇
- Persistent symptoms after treatment (possible resistant strain): cultures with sensitivities; continue current anti-TB and consult; consider other aetiologies (i.e. malignancy, fungal); treat empirically for bacterial pneumonia (e.g. ceftriaxone and doxycycline)

Anti-TB treatment

- Initiate treatment per national TB control programme guidelines. See 🕮 Antibiotics p. 968 for possible regimen ◇

TB management in HIV+ patients

- If presentation suggests TB, initiate full anti-TB course even if sputum negative
- All HIV+ patients with TB should be on ARVs, typically initiated 2–12 weeks after beginning anti-TB treatment. Monitor closely for immune reconstitution syndrome
- Cotrimoxazole PCP prophylaxis for HIV+ TB patients

Critical documentation

Method of diagnosis, HIV status, any prior treatment, complete local reporting guidelines.

Disposition

Admit newly diagnosed patients. Educate all regarding Directly Observed Treatment (DOTS) programme.

132 Influenza and Severe Acute Respiratory Syndrome (SARS)

Influenza and SARS are viral respiratory tract infections that have ability to cross from animals to human hosts, highly effective person to person transmission, (allowing for rapid global spread), and the capacity to cause severe respiratory distress and mortality. Both occur year round in tropical locations with a higher incidence in winter in temperate latitudes.

The first five minutes

ABC, VS, O_2 for signs of respiratory distress.

Institute proper precautions:

- Gloves, N-95 respirator mask for all suspected SARS and for airway procedures with suspected influenza
- Standard surgical masks for routine care of influenza
- Isolate or mask suspected influenza/SARS patient (N95 masks are intended for brief use by providers and do not vent CO_2 well – do not place on patients)

History and physical examination

Key historical features

High risk features: extremes of age (< 2 or > 65 years), HIV+, pregnant, chronic cardiopulmonary disease, other comorbid diseases. Time course, travel, and ill contacts.

Signs and symptoms

- SARS: prolonged prodrome (3–7 days of fevers, malaise, headaches, myalgias) without respiratory symptoms (easy to misdiagnose as malaria); followed at 3+ days by lower RTI
- SARS: clinical diagnosis (WHO): high fever (> 38 °C), **AND** cough + difficulty breathing **AND** exposure to a person thought to have SARS or travel through an area with an ongoing outbreak **AND** CXR consistent with findings of respiratory distress syndrome
- Influenza: abrupt onset of fever, fatigue, headache, sore throat, rhinitis, dry cough
- In suspected influenza patients who improve and then worsen, suspect secondary bacterial pneumonia

Differential diagnosis

- Early prodromal stages: malaria, typhoid, viral haemorrhagic fevers
- Respiratory phase: atypical bacterial pneumonia, other severe viral respiratory infections

Investigations

- Definitive investigations are unlikely to be available in the clinical setting
- CXR: variable from normal to diffuse bilateral infiltrates. Infiltrates in peripheral lower and middle lung fields are common

Influenza:

- Rapid influenza testing may be available in some settings, though sensitivity of most tests is poor
- CXR for significant respiratory distress, although findings may be non-specific

Management

The goal of acute management is limitation of exposure for staff and other contacts, restoration of oxygenation, and supportive care.

General

- Antipyretics (paracetamol) for fever
- Supplemental O_2 for tachypnoea, respiratory distress, or O_2 sat below 90% ◇
- Mechanical ventilation with low protective strategies (low tidal volumes, tolerance of hypoxia) may be necessary ◈

Specific treatment

Both infections are viral, and antibiotics are not helpful. However, definitive exclusion of bacterial infection is usually impossible. Treat those who deteriorate after initial improvement for secondary bacterial pneumonia (covering *Staphylococcus*), and treat all patients with significant respiratory distress for bacterial pneumonia. Only effective supportive care has improved outcomes in SARS patients.

Patients with suspected influenza and mild-moderate symptoms should receive supportive care only unless they are high risk. Patients with *severe* symptoms or high risk characteristics and presenting within 48 hours of onset can be treated with oseltamivir ◈, although evidence of benefit is limited:

- Adults: 75 mg BID × 5d
- Children 1–12y: < 15 kg (30 mg dose), 15–23 kg (45 mg dose), 23–40 kg (60 mg dose), > 40 kg (75 mg dose) BID × 5d
- Children < 1y: 3 mg/kg BID × 5d

Critical documentation

Serial VS and O_2 sat. Report suspected cases of SARS to local health authorities.

Disposition

Admit all patients with significant co-morbidities or respiratory distress, and admit patients with SARS per local public health recommendations.

133 Varicella

Primary infection with varicella zoster virus (VZV) causes varicella (chickenpox). Herpes zoster (shingles) is caused by reactivation of latent VZV in dorsal root ganglia. Congenital varicella syndrome may develop in infants born to mothers with varicella infection in the first 20 weeks of gestation.

Varicella can present at any age, but commonly before age 10. VZV is highly contagious with person-to-person spread by airborne transmission or by direct contact with skin lesions. Incubation period of primary varicella infection is 10–21 days.

The first five minutes

- ABC, VS, assess for respiratory involvement
- Exclude smallpox lesions by appearance (see below: chicken pox lesions should be in various stages of healing)

History and physical examination

Key historical features

- Vaccination status, and any history of similar illness
- Exposure to others with symptoms
- Timing and progression of lesions
- Chicken pox: prodrome of fever, headache and malaise 24-48 hours before onset of pruritic vesicular rash
- Herpes zoster: pain or tingling may precede vesicular rash

Signs and symptoms

- Chicken pox: crops of macules evolve into clear-fluid filled vesicles, which become cloudy and umbilicate, then crust over forming scabs:
  - » Lesions are in different stages on different parts of the body at the same time (distinguishes from smallpox lesions, all in the same stage)
  - » Age > 12 and immunocompromise leads to higher risk of complications
  - » Secondary infection of skin lesions (*S. aureus* or *S. pyogenes*)
  - » Pneumonia
- Herpes zoster: unilateral, dermatomal, painful, pruritic vesicles on a erythematous base

Differential diagnosis

- Chicken pox: smallpox, syphilis, impetigo, dermatitis herpetiformis
- Herpes zoster: contact dermatitis, coxsackie infection

Investigations

Diagnosis is clinical. CXR for respiratory findings.

Management

The goal of acute management is symptom reduction and complication monitoring.

- Symptomatic treatment: paracetamol, calamine lotion, adequate fluid intake
- If admitted, isolate in negative-pressure ventilation until lesions have crusted over. Patients are infectious from 1–2 days before onset of rash until all lesions have crusted over
- Acyclovir for hospitalised or complicated cases ◇. 20 mg/kg PO QD 5 days (maximum 3 200 mg/day) or 10 mg/kg IV TID 7–10 days for disseminated disease or immunocompromise
- Treat secondary bacterial infections with appropriate antibiotics

Prevention

Primary prevention: routine vaccination with live-attenuated vaccine reduces mortality by > 85%.

Post-exposure prophylactic options for non-immune individuals in health care settings:

- Varicella vaccine ◇: administer within 3–5 days of exposure in non-pregnant, immunocompetent and HIV-infected individuals > 12 months OR
- Varicella Zoster Immunoglobulin ◈: administer within 10 days of exposure, OR
- If vaccine and VZIG are unavailable, acyclovir 20 mg/kg QD for 7 days from presentation

Disposition

Admit patients with respiratory complications or diffuse suprainfection.

Patients with uncomplicated infection can be managed as outpatients. Children should not attend school or child-care facilities until lesions have crusted over. Avoid contact with immunocompromised household or community members until resolved.

134 Hepatitis

Hepatitis is injury to hepatocytes caused by inflammation or infection and characterised by liver enzyme elevations. Most patients with hepatitis are asymptomatic until 70–80% of the liver is injured. (See 🕮 Jaundice in adults p. 264 and Jaundice in children p. 262)

The first five minutes

- ABC, VS, O_2, IV
- Evaluate for AMS

History and physical examination

Key historical features

- Recent illness; prior liver disease**,** STI or blood transfusion
- Use of herbal remedies, medications, alcohol consumption, drug use

Signs and symptoms

- Fever, malaise, flu-like symptoms
- GI: nausea/vomiting/diarrhea, RUQ abdominal pain**,** anorexia
- Jaundice (look under tongue), icterus, dark urine
- Ascites

Possible causes and differential diagnosis

- Consider alcohol, medications (🕮 INH p. 680) and other toxins
- Viral haemorrhagic fevers (yellow fever) and other viral aetiology (including EBV, CMV)
- Amoebic liver abscess; and fasciola hepatica (liver fluke), migratory stage
- Autoimmune liver disease
- HELLP Syndrome (pregnancy induced liver disease) (🕮 p. 482)
- Hepatocellular carcinoma

• Cholangitis (🕮 p. 244)
• Leptospirosis

Investigations

• US to assess liver, spleen, bile ducts and gallbladder ◇
• Labs: CBC, BUN/Cr, AST, ALT, LDH, bilirubin (direct and indirect), alkaline phosphatase ◇; PT/PTT ◈. Serial LFTs and coags until peaked
• Viral hepatitis panels (A, B, C, E) for prior, acute and chronic infection ◈:
  » A: anti-A IgM = acute infection; anti-A IgG = prior infection, immunity
  » B: HBsAg (Hep B surface antigen) or anti-HBc IgM (IgM antibody to Hep B core antigen) = acute infection; anti-HBs (antibody to surface antigen) = vaccinated or > 3 months after prior infection, immune
  » C: HCV RNA PCR and ELISA
  » E: anti HEV (anti-Hep E antibody) IgM = acute infection

Table 134.1 Patterns of liver enzyme elevations and complimentary liver tests

	Peak ALT x upper limit of normal	AST/ALT ratio	Bilirubin peak	Prothrombin time (s)
Viral hepatitis	10–40	< 1	< 15	< 3
Alcoholic hepatitis	2–8	> 2	< 15	1–3
Toxic/drug /herbal	> 40	> 1 early	< 10–15	> 5
Leptospirosis	< 5	~ 1	> 15	1–2
Yellow fever	> 100	~ 1	> 10	
Ischaemic injury	> 40	> 1 early	> 5	> 5

Management

The goal of acute management is to maintain haemodynamic status and manage bleeding complications.
• Hep A, Hep B and yellow fever are major causes of fulminant hepatitis
• Treatment generally supportive: monitor electrolytes ◇, control bleeding; aggressive hydration for shock; otherwise cautious hydration to avoid fluid overload
• If paracetamol overdose suspected, treat within 8 h with N-acetyl cysteine (🕮 p. 655)
• If leptospirosis is suspected treat with ceftriaxone
• Monitor for: liver failure (confusion, encephalopathy, elevated PT/PTT); portal hypertension (ascites and /or varices); sepsis; elevated ICP; acute kidney injury or multi-organ failure

Disposition

• Admit all patients with climbing LFTs or abnormal coags, and with fulminant hepatitis to ICU

135 Typhoid and cholera

• **Typhoid:**also called enteric fever. Systemic illness caused by gram-negative bacteria *Salmonella enterica* (*S. typhi* and *S. paratyphi* serotypes); characterised by sustained fever and abdominal symptoms (diarrhoea in ~⅔ and more common in children; constipation in ~⅓). Faecal-oral transmission.
• **Cholera:**diarrhoeal illness caused by infection with gram-negative bacterium *Vibrio cholerae*. Profuse watery diarrhoea and vomiting leading to severe dehydration, rapid deterioration, and death in untreated patients. Faecal-oral transmission.

The first five minutes

• Transfer patient to isolation room and use contact precautions
• ABCs, VS, IV access. IVF for dehydration; glucose

History and physical examination

Typhoid

Key historical features

• Incubation period is 1–3 weeks; ask about ill contacts
• Non-specific history of febrile illness with abdominal pain

Signs and symptoms

In its classic form, untreated typhoid progresses through three phases, each lasting about one week:

1. Fever and bacteraemia with relative bradycardia when febrile (pulse-temperature dissociation)
2. Abdominal pain; 30–45% have ‘rose spots’ (pale pink/salmon macules on the trunk and abdomen)
3. Hepatosplenomegaly, GI bleeding, possible perforation due to ileocecal lymphatic hyperplasia of Peyer’s patches. Overall rate of perforation in untreated typhoid is 10%, with lower rates in children and higher in adults

• Nausea, vomiting and malaise may occur in any phase
• Fever in 3rd week suggests perforation/peritonitis
• Severe typhoid may be associated with ‘typhoid encephalopathy’: AMS, delirium (classically ‘calm’), confusion. Occurs in up to 15–20% of cases, is equally common in adults and children, and confers worse prognosis

Cholera

Key historical features

• Any known exposure to cholera, to endemic area or outbreak zone
• May have shellfish ingestion history

WHO standard clinical case definition

Suspect cholera when:

• In an area where the disease is not known to be present, a patient ≥ 5 years develops severe volume depletion or dies from acute watery diarrhoea
• In an area where there is a cholera epidemic, a patient ≥ 5 years develops acute watery diarrhoea, with or without vomiting

Signs and symptoms

• Classically, sudden onset relatively painless profuse, watery diarrhoea and vomiting, though onset may also be gradual. There may be abdominal cramping with diarrhoea but should not have pain otherwise. May be mild (appears as simple gastroenteritis) to severe. Fever uncommon
• Severe dehydration may develop within hours. Signs include: hypotension, tachycardia, lethargy, faint pulses, dry mucous membranes, oliguria, sunken eyes, slow skin pinch

Investigations

• Labs: CBC, renal function, electrolytes, LFTs, stool and blood cultures ◇
• Imaging: upright CXR if possible ileus or perforation

Management

The goal of acute management is hydration, early diagnosis, and institution of infection control measures. For cholera, significant mortality decrease when hydration status restored within first four hours and maintained.

Acute therapy

• Antipyretic: paracetamol as needed. Avoid non-steroidals in severe dehydration or acute kidney injury

• Antiemetic: odansetron, promethazine, prochlorperazine, metoclopramide, as needed

Typhoid

• IV fluids: bolus NS 20 ml/kg, reassess VS, urine output, hydration status
• Steroids: severe disease or encephalopathy only
• Dexamethasone 3 mg/kg first dose, then 1 mg/kg Q6h for 48 hours
• Antibiotics: refer to CDC/WHO current recommendations due to changing resistance patterns. Multi-drug resistant strains documented in Africa
  » Severe: ceftriaxone 100 mg/kg (max 4 gm) QD × 14 days
  » Uncomplicated: max dose fluoroquinolone × 7 days; preferably newer (gatifloxacin, gemfloxicin, moxifloxacin); OR if FQ resistance: azithromycin 10–20 mg/kg/day (max 1 gm) × 7 days
• If signs or symptoms of peritonitis: see 🕮Acute abdomen p. 748 consult surgery

Cholera

Mainstay of treatment is rehydration. Initial resuscitation:
• Severe dehydration/shock: IV crystalloids (Lactated Ringer's if available) 30 ml/kg over 30 mins followed by 70 ml/kg over 2–3 hours
  » Transition to ORS when perfusion and mental status improved
• Some dehydration: oral rehydration salts (ORS) in 4 hours
  » < 4 mo (< 5 kg): 200–400 ml
  » 4–11 mo (5–7.9 kg): 400–600 ml
  » 1–2 y (8–10.9 kg): 600–800 ml
  » 2–4 y (11–15.9 kg): 800–1 200 ml
  » 5–14 y (16–29.9 kg): 1 200–2 200 ml
  » > 14 y (≥ 30 kg): 2 200–4 000 ml
• No dehydration: oral rehydration salts (ORS)
  » Children < 2 y: 50–100 ml, up to 500 ml/day
  » Children 2–9 y: 100–200 ml, up to 1 000 ml/day
  » Patients > 9 y: 2 000 ml/day

Continued resuscitation:
• Strict in/out records. Monitor VS, radial pulse, capillary refill time. Increase rehydration rate if signs dehydration or shock
• Replace all volume lost in diarrhoea/vomit. One glass ORS for every diarrhoeal stool in adults, and ORS volume equal to diarrhoeal volume in children

Antibiotics are an adjunct to fluid resuscitation and reduce duration, and bacterial excretion, but need not be given emergently. Begin as soon as patient able to take oral medication. Refer to updated CDC or WHO recommendations; fluoroquinolone and tetracycline resistance documented in Africa:
• Pregnant or nursing women and children: azithromycin 20 mg/kg (max 1 gm) PO × 1 or erythromycin 12.5 mg/kg Q6h × 3 days
• Adults: azithromycin or doxycycline 300 mg PO × 1 or tetracycline 500 mg Q6h × 3 days

Cholera control:
• Management in treatment centres with strict isolation and sanitation; sufficient pre-stocked medical supplies; hygiene, food safety practices, public education

Critical documentation

Serial VS, document complications; results of testing; initial management note, including antimicrobial given; details of fluid resuscitation and clinical response.

Disposition

Admit severe typhoid to ICU or the highest level of care available. Patients with suspected ileal perforation: urgent

transfer to OT. Admit all patients with confirmed cholera in isolation wards/camp. Cholera treatment requires intensive hydration and close care; with appropriate rehydration, mortality is < 1%.

136 Rabies

Rabies is a vaccine-preventable viral disease of mammals most often transmitted by the saliva of an infected animal. Between 30 000 and 70 000 people die of rabies each year. This chapter addresses prophylaxis in acute bites, and management of the patient with clinical signs of rabies.

Presentation for acute bites

The first five minutes

Manage bite wound (📖 p. 220).

Key historical features

Type of animal, health/behaviour of animal (unprovoked attacks concerning), animal and patient vaccination status, current location of animal, and possibility of observing animal for 10 days, as this may allow later discontinuation of vaccine therapy.

Signs and symptoms

The combination of any systemic symptoms with complaints of numbness, tingling, itching from a bite wound is concerning for rabies.

Management to prevent rabies

The goal of acute management is early aggressive wound care, and rabies prophylaxis. Administer rabies vaccine/immunoglobulin as soon as possible as risk of infection increases over hours after exposure.

Wound care

Proper wound care significantly reduces risk of infection. Irrigate with copious water, scrub with dilute soap solution. Do not close wound. Give tetanus prophylaxis.

Rabies prophylaxis

There is no cure once a patient develops clinical signs of rabies. Therefore, all patients at risk should be treated as soon as possible with immunoglobulin (RIG) and rabies vaccine (RV) ◈. If RIG not available, clean wounds, give RV and refer. If RIG and RV unavailable, clean wounds immediately and refer. Since prolonged rabies incubation periods are possible, patients who present even months after having been bitten should be treated with RIG and RV.

RIG administration:
- Human rabies immunoglobulin (HRIG, 20 IU/kg) or equine rabies immunoglobulin (ERIG 40 IU/kg)
- Carefully infiltrate RIG at the wound site
- Inject IM any remaining RIG at a site distant from the site of RV inoculation
- If RIG cannot be given immediately, it should still be given up to 7 days after starting RV

RV administration:
- For the following use Purified VERO Cell Culture Rabies Vaccine (PVRV). One IM dose contains at least 2.5 IU in 0.5 ml
- Injected IM in deltoid in adults or anterolateral thigh in young children. Never use gluteal area (buttock) as fat deposits may interfere with uptake
- The 2-1-1 intramuscular regime:
  - Induces early antibody response; strongly recommended if RIG unavailable
  - Day 0 (1 dose in R arm + 1 dose in L arm), Day 7 (1 dose), Day 21 (1 dose)

- Alternative 2-site intradermal regime:
  - » This uses intradermal doses of 0.1ml (i.e. 1/5th of IM dose) of PVRV
  - » One dose into each of 2 sites, left and right deltoid, on days 0, 3 and 7; then one dose into 1 site, deltoid, on days 28 and 90
- Previously vaccinated persons (patients known to have previously received full rabies vaccination in < 3 years):
  - » One IM dose on days 0 and 3
  - » If vaccinated > 3 years earlier or incompletely, treat as unvaccinated

Critical documentation

Status of animal and observation plan, time and location of bite, time of administration of therapies, reporting per local guidelines.

Disposition

Admit severe or infected bite wounds (📖 p. 220).

Presentation for acute rabies

The first five minutes

Manage agitation to keep patient and caregivers safe.

Key historical features

Any known bites or possible exposure. Ask about animals behaving unusually or unexpected animal deaths. Incubation period is usually 1–3 months, but may range from weeks to years (90% < 1 year).

Signs and symptoms (usually progress over 1–2 weeks)

Prodrome (days to one week):

- Pain/paraesthesia at bite site with flu-like illness (fever, headache, N/V/D, and general weakness or discomfort)

Acute neurologic phase (days to one week):

- 80% have encephalitic form: anxiety, confusion, agitation, hallucinations, hyper-salivation, difficulty swallowing, and hydrophobia (fear of water) or aerophobia (fear of air or draft – administration of O_2 may cause laryngeal spasm, choking)
- 20% have paralytic form that may mimic GBS with limited and late cerebral involvement

Coma phase:

- Begins ~10d after symptom onset; death usually occurs within two weeks

Management

The goal of acute management is palliation, including pain control and sedation. Benzodiazepines, opioids, and ketamine may be useful agents.

Differential diagnosis

Non-specific viral illness; other encephalitis, toxic or metabolic encephalopathy.

Investigation

Diagnosis is usually clinical. In symptomatic patients, serum, saliva, CSF, and skin samples can be tested for viral antibodies, antigen, and RNA ◈ to confirm diagnosis.

Disposition

Admit for any clinical signs of rabies.

137 Schistosomiasis

Schistosomiasis is a waterborne parasitic disease caused by trematode flatworms. African species include *Schistosoma mansoni* (intestinal schistosomiasis) and *Schistosoma haematobium* (urinary schistosomiasis). Fresh water snails are the carriers of the infectious parasite larvae (cercariae), which penetrate the skin of people in water.

The first five minutes

- ABC, VS, O_2

Stages of infection

Schistosomiasis has three stages:

- **Cercarial dermatitis (swimmer's itch)**: due to entry of cercariae into skin, intensely pruritic papular eruption that occurs within hours to days of exposure and can last more than a week
- **Acute schistosomiasis (Katayama syndrome):** a systemic hypersensitivity reaction to migrating parasites, usually occurs 2–8 weeks after exposure
- **Chronic schistosomiasis:** granulomatous inflammation and fibrosis at site of parasitosis, occurs years after exposure

History and physical examination

Key historical features

High index of suspicion required as the acute illness may mimic viral, bacterial, or malaria infections. Inquire about drinking or physical contact with fresh water in endemic areas (while infections in other species can occur via salt water, human schistosomiasis is associated with fresh water). Chronic schistosomiasis can present months to years after infection; a lifelong history is essential.

Signs and symptoms

Acute schistosomiasis: occurs 2–8 weeks after contact with infested water.

- General features: rash (urticarial or papular) on exposed skin, fever, lethargy, malaise, cough, abdominal tenderness, headache, right upper quadrant pain
- Site specific features:
  - **Intestinal schistosomiasis:** fatigue, abdominal pain, diarrhoea or bloody diarrhoea, anorexia
  - **Urinary schistosomiasis:** dysuria, lower abdominal pain, urinary frequency, terminal haematuria, infertility

Chronic schistosomiasis: occurs years after initial exposure. Symptoms are a result of the host immune response to schistosoma eggs. Insidious onset with hepatosplenic, cardiopulmonary, and ectopic site (CNS) manifestations.

Investigations

- Labs: stool and urine analysis for schistosoma eggs ◇; schistosoma ELISA, urine PCR ◈
- Imaging:
  - XR KUB ◇: may show calcification of the bladder wall
  - US ◇: see www.who.int for WHO US criteria for likelihood and severity of schistosomiasis
    - Intestinal schistosomiasis: score based on parenchymal appearance, presence or absence of periportal infiltration, and evidence of portal vein enlargement
    - Urogenital schistosomiasis: score based on bladder thickening, calcification, irregularity, signs of hydronephrosis or hydroureter, and presence of prostatic or pelvic inflammation indicating advanced disease
  - Echocardiography to evaluate pulmonary hypertension and cor pulmonale ◇
- CT or MRI: to evaluate CNS and periportal disease ◈

Management

The goal of acute management is to eradicate the infectious agent and prevent the inflammatory complications of chronic illness.

- Medical treatment:
  - » Praziquantel PO 20 mg/kg TID for 24 hours. *Contraindicated in pregnancy* OR
  - » Oxamniquine PO 20 mg/kg single dose. *Contraindicated in pregnancy* OR
  - » Metrifonate PO 5 mg/kg three doses at two week intervals
- **Steroids** for symptoms of inflammation and patients with CNS manifestations:
  - » Prednisolone 20 mg PO BID for five days
- **Surgical treatment**: patients with complications such as tumour masses and granulomas of urinary bladder and lungs require surgical consultation ◇

Critical documentation

Suspected exposure. All investigations and results. Plan for antimicrobial treatment and follow-up.

Disposition

Admit patients with complications; consultation to medicine, surgery or urology as needed. All patients with suspected or confirmed uncomplicated schistosomiasis should be given treatment and outpatient follow-up.

138 Parasitic infections in the GI tract

Parasitic infections of the GI tract are very common and frequently asymptomatic. They may result in serious medical or surgical complications with emergency presentations.

The first five minutes

If clinical features suggest bowel obstruction or peritonitis: ABC, IV, nil by mouth**.**

History and physical examination

Key historical features

- Epsidoic diarrhoea, vomiting
- Passage of worms in stools or vomitus, perianal pruritis, weight loss

Signs and symptoms

- Intestinal infection: colitis (bloody dysenteric stools, abdominal pain, fever), bowel obstruction, biliary obstruction, peritonitis, anaemia
- Extra-intestinal disease: abscess or cyst formation (liver, lungs, pericardium, central nervous system, eye, skin, genitourinary tract), itchy papular rash, haematuria/dysuria, cough and fever, convulsions

Differential diagnosis

- Gastroenteritis, bacterial dysentery, urinary tract infection
- Malnutrition, HIV infection, TB, malignancy
- Other causes of bowel or biliary obstruction or peritonitis

Investigations

- Labs: CBC (Hgb, ecosinophil count), stool microscopy (ova, cysts, parasites) ◇; serological studies ◈

Management

The goal of acute management is rehydration and eradication of parasites to prevent GI complications and chronic anaemia.

- Diagnostic confirmation not always available/practical for intestinal nematodal (roundworm) or cestodal (tapeworm) infections
- Infection with multiple species is common. Empirical treatment with albendazole or mebendazole if unknown species

Disposition

Admit for:

- Severe diarrhoea and vomiting for rehydration/monitoring, particularly children
- Complicated disease (including any extra-intestinal disease manifestations)

Infection	Drug/s of choice	Adult dosage	Paediatric dosage
Amoebiasis (*Entamoeba histolytica*)	Metronidazole	500–750 mg PO TID x 7–10d	35–50 mg/kg/d PO in 3 doses x 7–10d
Balantidiasis (*Balantidium coli*)	Adults: tetracycline	500 mg PO Q6h x 10d	40 mg/kg/d (max 2g) PO in 4 doses x 10d
	Children < 8 yrs: metronidazole	500–750 mg PO TID x 5d	35–50 mg/kg/d PO in 3 doses x 5d
Cryptosporidiosis (*Cryptosporidium parvum/hominis*)	Nitazoxanide	500 mg PO BID x 3d	1–3 yrs: 100 mg PO bid x 3d 4–11 yrs: 200 mg PO bid x 3d > 12 yrs: 500 mg PO bid x 3d
Cyclosporiasis & cystoisosporiasis (*Cyclospora cayetanensis & Cystoisospora belli*)	Trimethoprim / sulfamethoxazole	TMP 160 mg/SMX 800 mg (1 DS tab) PO BID x 7–10d	TMP 10 mg/kg/SMX 50 mg/kg/d PO in 2 doses x 7–10d
Giardiasis (*Giardia intestinalis*)	Metronidazole	250 mg PO TID x 5–7d	15 mg/kg/d PO in 3 doses x 5–7d
Paragonimiasis (*Paragonimus* trematodes causing lung fluke disease)	Praziquantel	75 mg/kg/d in 3 doses x 2d	75 mg/kg/d in 3 doses x 2d
Round worms (Nematodes)			
Ascaris, Ancylostoma, Necator	Albendazole OR Mebendazole	400 mg PO once 100 mg PO BID x 3d	400 mg PO once 100 mg PO BID x 3d
Enterobius	Repeat either of above doses after 2 wk for *Enterobius*		
Strongyloidiasis	Albendazole	400 mg PO BID x 7d	400 mg PO BID x 7d
Toxocariasis	Albendazole OR mebendazole	400 mg PO BID x 5d 100–200 mg PO BID x 5d	400 mg PO BID x 5d 100–200 mg PO BID x 5d
Trichuris	Albendazole OR mebendazole	400 mg PO x 3d 100 mg PO BID x 3d	400 mg PO x 3d 100 mg PO BID x 3d
Tapeworms (adult intestinal stage)			
Diphyllobothrium, Dipylidium, Taenia species	Praziquantel	5–10 mg/kg PO once	5–10 mg/kg PO once
Hymenolepis	Praziquantel	25 mg/kg PO once	25 mg/kg PO once
Schistosomiasis	Praziquantel	40 mg/kg/d PO in 1–2 doses x 1d *S. japonicum* or *mekongi*: 60 mg/kg/d PO in 2–3 doses x 1d	40 mg/kg/d PO in 2 doses x 1 d

139 Sexually transmitted infections

STIs are a group of diseases caused by bacteria, viruses, and parasites that are spread through sexual contact. These diseases are common, spread rapidly, cause serious illnesses, can affect pregnancy and child birth, are linked to the spread of HIV/AIDS and can have serious long term effects on health.

Approach to diagnosis and management

Diagnosis

Most experts recommend a ‘syndromic’ approach, which categorises STIs into clinical syndromes (Table 139.1) and targets treatment to cover all causative agents. This approach provides a reliable guide to initiate treatment even in the absence of laboratory diagnosis.

Management

The goal of acute management is antimicrobial therapy, contact tracing, and counselling to limit further exposure.

- STIs significantly increase the risk HIV transmission. It is important to counsel patients and test for HIV infection
- Consider testing or empiric treatment for syphilis depending upon local prevalence
- Always treat both chlamydia and gonorrhoea. Co-infection is extremely common
- Pelvic inflammatory disease can be a difficult diagnosis and complicated treatment. Have a low threshold to diagnose, treat, and hospitalise. Patients discharged should be reviewed in three days
- Counsel using the 4Cs

> **Counselling:** empathise with patient, dialogue and discuss the other 3Cs
> **Compliance:** patient should take the full course of medication and not share or keep it
> **Condoms:** proper condom use is essential. Provide condoms, explain and demonstrate their proper use
> **Contact tracing:** patient should tell all sexual partners within the past 60 days (90 days for genital ulcer) to seek medical attention

Table 139.1 STI Syndromes and empiric treatment

Vaginal discharge without systemic symptoms
Out-patient treatment Ceftriaxone 250 mg IM ×1 (gonorrhoea) AND EITHER azithromycin 1 g PO × 1 OR doxycycline 100 mg BID × 7 days (chlamydia) (doxycycline contraindicated in pregnancy) AND Metronidazole 2 g PO x1 for trichomonas, gardnerella If curd-like discharge, erythema, or itching/excoriations, ADD fluconazole 150 mg PO × 1 for candida
Clinical notes • *Neisseria gonorrhoea*: greenish yellow discharge oozing from endocervix. Presents with dysuria, frequency. Complications include tubal blockage → infertility or ectopic pregnancy, Bartholin’s abscess, disseminated infection, ophthalmia neonatorum. • *Chlamydia trachomatis*: scanty muco-purulent or purulent discharge. Minimal or no symptoms. May have painful micturition and vulvo-vaginal itching. Complications include ophthalmia neonatorum, pelvic infection → tubal blockage and infertility or ectopic pregnancy. • Bacterial vaginosis *(Gardnerella)*: profuse foul-smelling and homogenous grey-white discharge adherent to vaginal wall. • *Trichomonas vaginalis*: frothy, profuse, greenish yellow, foul smelling discharge. May be associated with vulvo-vaginal itching; swollen Bartholin glands. • *Candida*: white, curd-like discharge involving the vaginal wall and cervix. Presents with vaginal itching; inflamed, swollen vulva if severe.
Vaginal discharge with lower abdominal pain or cervical motion tenderness **(PID: pelvic inflammatory disease)**

Out-patient treatment
If not pregnant, afebrile, able to take PO, and well-appearing:
Ceftriaxone 250 mg IM x1
AND
Doxycycline 100 mg BID × 14 days
AND
Metronidazole 500 mg BID × 14 days
In-patient treatment
If pregnant, febrile, ill-appearing, tubo-ovarian abscess or palpable adnexal mass, unable to take PO, nausea and vomiting, no improvement on oral medications, or requires monitoring for possible surgical emergency (e.g. appendicitis), continue therapy for at least 2 days after patient improvement, then complete course with doxycycline 100 mg BID × 14 days as above. If tubo-ovarian abscess, discharge on clindamycin or metronidazole in addition to doxycycline.
Ceftriaxone 250 mg IM daily
AND
Doxycycline 100 mg PO/IV BID
AND
EITHER metronidazole 500 mg PO/IV BID OR chloramphenicol 500 mg PO/IV Q6h
IF PREGNANT OR PENICILLIN-ALLERGIC:
Clindamycin 900 mg IV TID AND gentamicin 5 mg/kg IV daily

Clinical notes
- Pelvic abscess, tubal blockage → infertility, ectopic pregnancy, septicaemia bacteremia.
- Gonorrhoea has increasing quinolone resistance.

Vaginal discharge after recent instrumentation (surgical procedure, abortion)

Regimen as above for PID.
Consider MRSA coverage (e.g. vancomycin) based on local prevalence.

Urethral discharge syndrome (gonorrhoea, chlamydia)

Treatment
Ceftriaxone 250 mg IM × 1
AND
EITHER azithromycin 1 g PO x1 OR doxycycline 100 mg PO BID × 7 days
If recurrent or no improvement in 7 days, consider metronidazole 2 gm PO x1 for trichomonas

Clinical notes
- *Neisseria gonorrhoea*: purulent discharge from the urethra. May cause painful and frequent micturition with or without testicular pain. Complications include urethral stricture, epididymo-orchitis, prostatitis, infertility, disseminated infection.
- *Chlamydia trachomatis*: scanty mucoid or serous discharge, greatest in the morning. May present with urethral irritation; painful, frequent micturition. Complications include epididymo-orchitis, infertility.

Painful scrotum (epididymo-orchitis)

Treatment
Doxycycline 100 mg BID × 10 days
AND
Ceftriaxone 250 mg IM x1 dose
If risk for enteric organisms
ADD ciprofloxacin 500 mg PO BID × 10 days

Clinical notes
- Organisms include *N. gonorrhoea*, *Chlamydia trachomatis*. Men over 35yrs, men practicing insertive anal sex, those with urinary tract instrumentation at risk for enteric organisms. If no improvement at 1 week, consider antibiotic resistance, brucella, TB, mumps, or other atypical aetiology.

Genital ulcer disease

Treatment
Empirically treat syphilis: benzathine penicillin 2.4 M units IM x1
ADD acyclovir if herpes suspected:
1st episode: acyclovir 400 mg TID × 7 days
Recurrent episode: 400 mg TID × 5 days
HIV+: 400 mg 5x daily until resolution and consider genital ulcer co-infection.
If pregnant with recurrent HSV, treat neonate empirically 10 mg/kg IV TID × 21 days
ADD empiric antibiotics for other ulcer diseases based on local prevalence
LGV: doxycycline 100 mg BID × 14 days OR erythromycin 500 mg q6h × 14 days
CHANCROID: ciprofloxacin 500 mg BID × 3 days OR azithromycin 1 gm PO × 1
OR erythromycin 500 mg Q6h × 7 days OR ceftriaxone 250 mg IM × 1
GRANULOMA INGUINALE: azithromycin 1 gm PO x1, then 500 mg daily until epithelialised OR doxycycline 100 mg PO BID.

ADD gentamicin 5 mg/kg/day if HIV+

Clinical notes

- *Herpes simplex virus*: multiple shallow, painful ulcers; start as grouped vesicles. May have tender lymphadenopathy. Complications include infection of the newborn at birth.
- *Treponema pallidum* (syphilis): single, *painless*, relatively clean ulcers without pus. Primary, secondary, tertiary stages; may be latent for years.
- *Haemophilus ducreyi* (chancroid): multiple, soft, deep, tender ulcers with purulent discharge. Painful, fluctuant lymphadenopathy, may result in genital disfiguration, lymphatic obstruction.
- *Lymphogranuloma venereum* (LGV): single, small, transient ulcers. May have matted lymphadenopathy, fistula and stricture formation, tissue destruction.
- *Granuloma inguinale* (GI; Donovan bacilli): large, beefy ulcers; rare lymphadenopathy; pseudo-buboes.

Genital warts

Treatment

Patient-applied: podofilox 0.5% BID × 3 days, then none x4 days, then repeat cycle x4 as needed.
Surgical: cryotherapy, electrosurgery, surgical removal.

Clinical notes

- *Condyloma acuminata* (human papilloma virus, HPV): cauliflower-like warts, single or multiple, over the genitalia and perineum. May have pain and/or bleeding on coitus or touch. At risk for secondary infection, cancers of the penis, cervix, vulva.

140 Botulism

Caused by a neurotoxin produced by the bacterium *Clostridium botulinum*, botulism is a neuroparalytic syndrome that is potentially life threatening. The disease is acquired by either ingestion or inhalation of spores, or by wound contamination (including contamination during IV drug use).

Botulism can divided into four categories, identified by how the disease is acquired:

- **Food borne botulism**: ingestion of pre-formed botulinum toxin in contaminated food
- **Infant or adult botulism**: spores are ingested (honey is a common source in infants), allowing *C. botulinum* to colonise the digestive tract and release toxin
- **Wound botulism**: neurotoxin is released by *C. botulinum* that has colonised a wound, including those associated with IV drug use
- **Inhalational botulism**: occurs when the toxin itself is aerosolised

The first five minutes

ABC, O_2, IV; focused neuro exam, including strength of respiratory effort and careful CN exam for ability to protect airway.

History and physical examination

Key historical features

- Diplopia, dysarthria, dysphagia are classic complaints
- In general, there should be *no history of fever* (except for possibly wound botulism)

Signs and symptoms

- Mentation and sensation should be normal
- Speech may be dysarthric
- Ophthalmoplegia and dilated pupils → visual complaints
- Weakness of muscle groups in arms and legs (late finding)
- Urinary retention (late finding)

Differential diagnosis

Brainstem stroke, myasthenia gravis, GBS, acute inflammatory demyelinating polyradiculopathy, Lambert-Eaton Syndrome, polio.

Investigations

- In many settings, this will be a clinical diagnosis
- Lab confirmation: detection of *C. botulinum* in the stool or of botulinum toxin in the patient's serum, stool, or wound ◈
- Maximum/negative inspiratory force testing ◈
- Electrophysiologic testing ◈ can provide early presumptive evidence of botulism when clinical picture suggestive, but stool cultures and toxin bioassays negative

Management

The goal of acute management is respiratory support, and early antitoxin.

Supportive

- Careful frequent re-evaluation of respiratory status
- Early transfer of suspected cases to a centre with capability for mechanical ventilation, as intubation for airway protection and ventilatory support is often needed ◈
- Supplemental nutrition and DVT prophylaxis during admission ◇

Specific treatment

- Give a test dose of equine antitoxin, where available. If no adverse reaction, give full dose (do not wait for lab results) ◈
- Infants less than a year should be treated only with human derived botulism immune globulin ◈
- Do NOT administer antibiotics in suspected cases of infant botulism or adult with suspected gastrointestinal botulism. Antibiotics will cause increased release of toxin from lysis of intra-luminal *C. botulinum*
- If food borne botulism is suspected, consider GI decontamination with laxatives or enemas unless patient has an ileus ◇
- Perform wide and aggressive surgical debridement of *any* wound that is suspected to harbour *C. botulinum* regardless of how benign it appears ◇

Critical documentation

- Serial VS, O_2 sat, neurologic exams, results of any respiratory testing

Disposition

Admit all patients with suspected botulism. Referral to a centre with capacity for mechanical ventilation is recommended.

141 Tetanus

A toxin (tetanospasmin) mediated disease caused by *Clostridium tetani*, a gram positive anaerobe; characterised by painful generalised muscle spasms and possible respiratory arrest. *C. tetani* is ubiquitous in nature and exposure occurs through open wounds. This disease is prevalent in Africa, with mortality reaching 60% in some countries.

To PREVENT tetanus: irrigate wounds well and treat patients unimmunised within past five years with tetanus toxoid 0.5 ml IM**.** Consider adding tetanus immunoglobulin for contaminated wounds.

The first five minutes

ABC, VS, facemask O_2**,** IV

History and physical examination

Key historical features

Initial wound may be trivial or unnoticed (40%).
- Incubation period from 2–40 days
- Shorter incubation correlates with more severe disease
- All age groups affected; neonates exposed in regions where cow dung is commonly applied to umbilical cord stump

Signs and symptoms (vary with type)

Generalised tetanus

- Usually begins with trismus (lock jaw) and dysphagia
- Risus sardonicus – severe facial muscle spasms with sardonic 'smile'
- Facial palsy may be present
- Progresses to generalised muscular rigidity with arched back (opisthotonus)
- Autonomic instability (sweating, fever, tachycardia, salivation, arrhythmias, hyper- or hypotension)
- Lucid mental state (patient is aware of the muscle stiffness)
- May progress to respiratory arrest

Localised tetanus

- Rare form of tetanus where localised muscle spasms do not generalise

Cephalic tetanus

- Rare form of tetanus characterised by cranial nerve palsies and associated with a high mortality

Neonatal tetanus

- Secondary to poor umbilical hygiene and associated with high mortality
- 90% of cases occur between day of life 3–14
- Poor suckling and feeding due to trismus
- Muscle rigidity and spasm worse with stimulation

Differential diagnosis

- Other local jaw infection and trauma can cause trismus
- Hypocalcaemia
- Dystonic drug reactions, especially to phenothiazines
- Somatisation

Investigations

Diagnosis is usually clinical. Monitor creatinine kinase and renal function ◇.

Management

The goal of acute management is aggressive wound management, and early administration of vaccine/immunoglobulin. All patients MUST be monitored for respiratory arrest related to spasm.
- Debride and irrigate wound
- Avoid light and other stimuli which will worsen the spasms
- Treat spasms with IV diazepam, chlorpromozine and continuous magnesium infusion ◇
- Treat infection: metronidazole 500 mg TID, or penicillin G 2 million units QD ◇
- Neutralise toxin:

   - Human hyperimmune globulin (3 000–6 000 IU IM single dose) IM (neonates 500 IU IM single dose) OR equine anti-tetanus globulin (0–1 500 IU IM/IV) (give a pretest dose to avoid anaphylaxis)
   - Tetanus vaccine (3 doses, 2 weeks apart, if unvaccinated)
- Respiratory: intubation ◇ for signs of respiratory insufficiency or inability to maintain oxygenation. Paralyse to assist in mechanical ventilation
- Autonomic instability: control with short acting beta blocker or clonidine; if severe add atropine or morphine ◇

Critical documentation

Wound management, toxin neutralisation strategy.

Disposition

All patients need close monitoring for respiratory arrest during acute spasm. Admit to ICU if possible.

142 Infectious and post-infectious spinal disease

Infectious or post-infectious inflammation or destruction of the vertebral column, disc space, canal, cord (myelitis) and adjacent soft tissues.

The first five minutes

ABC, VS, O_2 if hypoxic, IV, monitor IVF for shock. Assess strength of respiratory effort.

History and physical examination

Key historical features

- Assess risk factors: post-surgical, immunocompromise, advanced age, malnutrition, cancer, environmental/travel exposures, trauma

Signs and symptoms

Table 142.1 Clinical manifestations and evaluation by aetiology

	Organisms	Clinical symptoms	Laboratory	Radiography
Vertebral osteomyelitis or bacterial epidural abscess	*S. aureus*, Enteric GNR, Streptococci Others: *P. aeruginosa*, *Candida*, Meliodosis, *E. histolytica* TB, brucellosis	Back pain, fever, chills, weight loss, soft tissue mass (abscess); muscle spasms, incontinence/ neurological deficits	◇: CBC, ESR/ CRP, ◈: blood culture, bone culture/AFB	Vertebral XR ◇: bony destruction (TB-Gibbus deformity) MRI, CT, or CT-guided aspirate ◈
Grey matter disease	Schistosomiasis: eggs/granulomas embolise venous plexus. Adult worms may migrate to cord. Polio, rabies, West Nile Virus	Acute flaccid paralysis Areflexia; eventual muscle atrophy Unusual sensory or autonomic (bowel/ bladder) disturbances	◇: CSF-nonspecific mononuclear pleocytosis; viral specific IgM or PCRs	MRI with gadolinium ◈
White matter disease	HSV, VZV (rash - associated) EBV, CMV (esp. if HIV+)	Transverse myelitis: 🕮 p. 456 Weakness, pain/ tingling, incontinence, autonomic disturbance		
Chronic viral cord infection	HIV, HTLV	Slow onset (months) HIV: varied, spastic paraparesis HTLV: LE weakness/ spastic gait, incontinence, impotence	HIV/CD4 testing ◇; serum/CSF viral panel ◈	None

GNR: Gram Negative Rods

Considerations on specific organisms

Brucella

- May have history of tending goats or cattle or drinking unboiled milk
- XR with lytic or blastic lesions (TB has mostly lytic); bony destruction rare

Polio

- For each clinical case there are many asymptomatic cases. Alert health officials

West Nile Virus

- Paralysis, including respiratory paralysis, can occur in isolation

Management

The goal of acute management is early antibiotics, control of inflammation and oedema to limit sequalae, and rapid identification of need for surgical decompression.

Table 142.2 Antimicrobials, by causative agent

Organism	Antimicrobial treatment
Empiric antibiotics for bacterial disease	Initial treatment: broad-spectrum coverage Vancomycin 15–20 mg/kg BID AND ceftriaxone 1–2 gm IV daily If concern for anaerobes (abdominal source), ADD metronidzole 500 mg Q6h If concern for *Pseudomonas*, replace empiric ceftriaxone with cefepime 2 gm BID OR ciprofloxacin 400 mg IV BID Continue IV antibiotics for at least 4–6 weeks, following inflammatory markers (ESR, CRP) for resolution. Transition to PO antibiotics and continue course for 8–12 weeks total Obtain cultures and narrow antibiosis accordingly
M. tuberculosis (Pott's disease)	Multi-drug therapy per national recommendations × 6–9 months. See 🕮 Antibiotics p. 966
Brucellosis	Doxycycline 100 mg PO/IV BID AND rifampin PO 600–900 mg daily AND ceftriaxone 1 gm IV daily

	Therapy ongoing for months. Consult specialist.
HSV/VZV	Acyclovir, 10 mg/kg IV TID for 14–21 days ◇
CMV	Ganciclovir 5 mg/kg BID × min 2 weeks, then maintenance dose. Foscarnet reserved for ganciclovir toxicity ◈. Consult specialist
EBV; enteroviruses	Consult specialist. Consider immunoglobulin, steroids, ganciclovir ◈
HIV	Supportive care, ARV
HTLV-1	Supportive care; corticosteroids may slow progression
Schistosomiasis	Praziquantel 20 mg/kg PO BID × 2–3 days ◇ Corticosteroids; consult specialist for duration and taper. Surgical decompression if cord compressive symptoms. May require multiple doses praziquantel to clear – re-evaluate at 6 weeks.

Neurosurgical care ◈

• Vertebral osteomyelitis: decompression, abscess drainage, or hardware to stabilise spine

Supportive care

• Physical therapy, rehabilitation

Critical documentation

Serial VS and neuro exams. Response to therapy.

Disposition

Admit all patients with a suspected spinal infection.

143 Kawasaki disease

Also known as mucocutaneous lymph node syndrome, a microvasculitis of unknown aetiology that is probably post-infectious. Peak incidence 1–2 years old**.** The major associated morbidity is coronary artery involvement: aneurysms occur in 20% of untreated patients and 5% of treated; higher peak and longer duration of fever are risk factors for coronary involvement. Aneurysms resolve in 60% after two years.

The first five minutes

• ABC, VS , facemask O_2, IV, cardiac monitor
• ECG

History and physical examination

Key historical features

Ask about recent febrile illness, rash, oral lesions or pain (poor feeding may result), swollen glands**.**

Physical signs

Fever (90% of cases); irritable and ill-appearing; red eyes and/or mouth with cracked lips (90%); polymorphous rash (70%). Evaluate for cardiac involvement (valvular murmurs, pericardial friction rub, arrhythmias, CCF).

Clinical course can be divided into three phases:

Acute phase (first 10 days) diagnostic criteria

Fever (abrupt onset of high fever ≥ 39˚C, persisting at least five days) AND
four or more of the following:

• Extremity changes:

  - Acute: erythema and oedema of hands and feet
  - Sub-acute: periungual peeling of toes and fingers
- Polymorphous exanthema
- Bilateral non-exudative conjunctivitis
- Lips and oral cavity changes: cracked lips, strawberry tongue, other erythema of oral cavity
- Cervical lymphadenopathy: > 1.5 cm, usually unilateral

Other associated symptoms may include hepatitis, aseptic meningitis, uveitis, arthritis, urethritis or gastro-enteritis. Incomplete/atypical Kawasaki may be diagnosed if full criteria are not met but other clinical signs and investigations support the diagnosis.

Subacute phase (11–25 days) diagnostic criteria

- Desquamation of toe and finger tips; rash, fever and lymphadenopathy disappear
- Thrombocytosis
- Cardiovascular involvement: congestive cardiac failure, myocarditis, pericarditis, endocarditis and coronary vasculitis

> Mortality and morbidity are due to cardiac complications such as arrhythmias, myocardial infarcts, coronary aneurysm rupture or cardiac failure.

Convalescent phase (> 25 days)

Coronary vasculitis healing with scars and stenosis. Infarcts mostly during first 6–8 weeks. Ends when ESR and platelet count return to normal.

Differential diagnosis

- Viral exanthema (measles, adenovirus, EBV, Parvovirus B19)
- Bacterial infections (scarlet fever, staphylococcal scalded skin syndrome, toxic shock syndrome or rickettsial disease)
- Drug hypersensitivity, Stevens-Johnson syndrome
- Rheumatoid arthritis
- Other vasculitis

Investigation

- Labs: CBC (elevated WBC, anaemia), ESR ⟐, albumin (low), CRP ⟐
- ECG ◇: ST segment, T and Q waves changes
- CXR ◇: cardiomegaly
- Echocardiography ⟐:
  - Assess myocardial function, valvular regurgitation, pericardial effusion
  - Assess coronary arteries for: irregularity and ectasia, dilatation and aneurysms, and thrombi

Management

The goal of acute management is rapid diagnosis, symptom relief, reduction of inflammation to minimise complications, and detection and treatment of existing cardiac complications.

Aspirin

- Acute phase: 80–100 mg/kg/day aspirin in 4 doses
- If afebrile for 72 hours reduce aspirin to 5 mg/kg/day
- If no or transient coronary abnormality discontinue aspirin after 6–8 weeks

Intravenous gamma globulin (IVIG) ◈

IVIG reduces the risk of coronary disease.

- 2 g/kg IVIG over 12 hours, if diagnosed within 10 days of onset. Repeat dose if fever persists or recurs. Risk for anaphylaxis: treat with steroids and antihistamines
- IVIG can still be given if present after 10 days of illness with ongoing inflammation

Steroids

- Consider methylprednisone if fever persists after two doses IVIG, but this is controversial ◈

Anticoagulation

- For giant aneurysms:
  - Aspirin 5 mg/kg aspirin long term AND
  - Warfarin (target INR 2–3) ◇ OR low-molecular-weight-heparin (target anti-factor Xa 0.5–1 u/ml) ◈

CCF therapy

- Treat with diuretics and afterload reducers as needed ◇

Critical documentation

Severity assessment, medication doses, timings, response to treatment.

Disposition

Admit all patients with suspected acute onset Kawasaki for IVIG; refer to cardiology ◈. Chronic Kawasaki with suspected cardiac complications: refer to cardiology ◈; follow up cardiology assessment 6–8 weeks.

144 Acute rheumatic fever

A multisystem disease caused by abnormal immunological response to Group A streptococcus infection, targeting the heart, joints, nervous system and skin. ARF follows an acute recurrent course usually beginning in childhood (5–15 years). Each episode aggravates heart valve damage and ARF is the most common cause of acquired valvular disease in the developing world.

The first five minutes

- ABC, VS, facemask O_2, cardiac monitor
- Place 2 large bore IV, start IV crystalloid bolus if the patient is in shock

History and physical examination

Key historical features

- Pharyngitis in the preceding 10 days to six weeks
- Fever; abdominal and chest pain
- Migratory polyarthritis involving large joints

Signs and symptoms

- Carditis: resting tachycardia; diffuse apical impulse (myocarditis); cardiac murmurs – usually left heart valves (endocarditis); pericardial friction rub (pericarditis); ± CCF (inflammatory myopathy)
- Polyarthritis: sequentially inflamed large joints
- Rheumatoid nodules: small non-tender subcutaneous nodules over the extensor surfaces of upper and lower limbs, back, occiput, scalp and ears

- Erythema marginatum: circular erythematous rash on trunk and limbs; comes and goes leaving hyperpigmented lines as it fades. Hard to see on darker skin
- Sydenham's chorea: asymmetrical purposeless movements and associated emotional lability and speech impediment (female predominance)

Diagnostic criteria

Diagnosis is based on revised Jones' criteria (Table 144.1) and requires two major criteria or one major and two minor criteria PLUS evidence of strep infection (i.e. positive throat culture or indirect evidence of strep presence). However, rigid use of Jones' criteria has been shown to lead to underdiagnosis; treat for high clinical suspicion when definitive studies not available.

Table 144.1

Criteria	Major	Minor
1	Carditis	Prolonged PR interval on ECG
2	Polyarthritis	Arthralgia*
3	Sydenham's chorea	Previous history of evidence of rheumatic fever
4	Erythema marginatum	Fever
5	Subcutaneous nodules	Acute phase reactants (ESR and CRP)

*NB: Arthralgia should not be counted as a minor manifestation when arthritis is counted as a major.

Differential diagnosis

Kawasaki disease, septic arthritis, bacterial sepsis, endocarditis, congenital heart disease**.**

Investigations

Assess clinical criteria and probability of recent strep infection

- Labs: CBC (↑WBC), ESR ◇; CRP, cardiac enzymes, throat swab for culture or ASOT and anti-DNase B or anti-hyaluronidase ◈
- ECG ◇: PR > 0.18 seconds
- Imaging: CXR ◇ (cardiomegaly); echo ◈

Management

The goal of acute management is symptomatic care and eradication of strep infection or carrier state to limit inflammatory complications.

  » Benzathine penicillin G, IMI once (600 000 units ≤ 30 kg, and 1 200 000 units ≥ 30 kg) OR penicillin V 250–500 mg Q6h for 10 days taken orally
  » Penicillin allergy: erythromycin 250–500 mg (children 6.25–12.5 mg/kg/dose) Q6h for 10 days
- Inflammatory symptoms and fever:
  » Aspirin 80–120 mg/kg/day in 4 divided doses OR (if contraindication ibuprofen 300–800 mg TID (children 10 mg/kg/dose Q6h)
- Chorea
  » Haloperidol 0.025 mg/kg/day in 2–3 divided doses, increase to 0.05 mg/kg/day
- Congestive cardiac failure: (📖 p. 104)
- Prevention of repeat attacks:
  » Benzathine benzylpenicillin (LA), IM every 21–28 days (< 30 kg 600 000 units; if > 30kg 1 200 000 units) OR Penicillin V 250 mg BID
  » Pen allergic: erythromycin 250 mg orally QD

Critical documentation

Presence of murmur, vital observations, diagnostic criteria.

Disposition

Admit for bed rest until sleeping pulse is normal and features of active disease resolve. Restrict physical activity for two weeks after disease activity ceases.

145 Viral haemorrhagic fevers

VHFs are aetiologically diverse viral illnesses that share clinical characteristics and pathophysiology related to microvascular damage and increased vascular permeability. Dengue is a VHF with distinct clinical qualities and is addressed in a separate chapter (📖 p. 374).

Suspect VHF in patients with any unexplained bleeding from mucous membranes (gums, nose or vagina), skin (puncture sites, petechiae), conjunctiva (red eyes due to swollen blood vessels), or GIT (vomiting blood; dark or bloody stools); or in any ill febrile patient who has had contacts with unexplained death, febrile illness, or bleeding within three weeks.

The first five minutes

Isolate any patient with suspected VHF, and institute strict contact and droplet precautions. Anyone in contact with the patient, specimens, or linens should wear a scrub suit, gown and apron, two pairs of gloves, mask, head cover, eyewear, and rubber boots.

- ABC, VS, facemask O_2, IV; cardiac monitor. Start IVF for shock

History and physical examination

Key historical features

- Contact with infected person or natural reservoir (e.g. African fruit bats, rodents). High risk occupations: miners, hunters. Ask about relatives/friends who died recently
- Incubation can be as short as a few days, and is usually < 3 weeks

Signs and symptoms

- Abrupt onset of high fever, severe headache and myalgias (similar to malaria)
- Fatigue, confusion, agitation, vomiting, abdominal pain, diarrhoea, skin rashes and bleeding (a LATE finding)
- Exam findings: hyperaemic conjunctiva, high temperature, mild hypotension, skin rashes, flushing, and petechial haemorrhages

Possible causes and differential diagnosis

Differential diagnosis: malaria, enteric fever, gram-negative bacterial septicaemia, staphylococcal or streptococcal toxic shock syndrome, meningococcemia, septicaemic plague, leptospirosis, viral hepatitis.

Investigations

Clinical diagnosis; labs confirm. Only a few African labs have the biosafety facilities to perform specific VHF tests.

- CBC (mild anaemia, thrombocytopaenia, and leukopaenia) ◇; elevated PT/PTT ◈
- LFTs ◇: in yellow fever and Lassa fever, elevations early
- Specific VHF testing ◈: IgM antibody, paired serologies (at 7 and 14–27 days after symptom onset), antigen-capture ELISA (avoid EDTA tubes), or PCR. Gold standard is viral identification in cell culture

Management

The goal of acute management is prevention of transmission, and supportive care (maintenance of fluid and electrolyte balance, and antimicrobials for secondary infections).

- Avoid all NSAIDs, aspirin, anticoagulants, and intramuscular injections
- Mechanical ventilation and dialysis as needed ◈

Specific therapy

- IV ribavirin in Lassa fever and Rift Valley Fever ◈. Load with 30 mg/kg (max 2 gm) IV, then: 16 mg/kg (max 1 gm) IV Q6h for 4 days, then 8 mg/kg (max 500 mg) IV TID for 6 days
- If no IV Ribavirin, then oral for 10 days:
  - » > 75 kg: load 2 g PO, then 600 mg/day PO BID
  - » < 75 kg: load 2 g PO, then 400 mg PO in am and 600 mg in pm

Critical documentation

Report suspected cases to health authorities.

Disposition

Admit all patients with suspected VHF to an isolation bed.

Table 145.1 Viruses that cause VHF outbreaks in Africa

Viral family and disease	Geographical areas
Arenaviridae: Lassa fever, Lujo virus	Lassa fever: West Africa (Sierra Leone, Guinea, Liberia, and Nigeria) Lujo virus: Zambia, South Africa
Bunyaviridae: Crimean-Congo Haemorrhagic Fever, haemorrhagic fever with renal syndrome, Rift Valley fever	Human RVF cases reported in Egypt, Madagascar, Mauritania, Kenya, Somalia, Tanzania, South Africa
Filoviridae: ebola virus and Marburg virus.	Ebola: DRC, Côte d'Ivoire, Gabon, Sudan, Uganda Marburg: Uganda, Kenya, DRC, Angola, Zimbabwe
Flaviviridae: yellow fever virus and dengue fever	Dengue: most countries Yellow fever: West, East and Central Africa

146 Dengue fever

Dengue is a widespread viral infection transmitted by *Aedes aegypti* mosquitos that affects 50 million people per year in over 100 countries. Large portions of Africa currently experience transmission, and even more have appropriate vectors and conditions for transmission (see http://www.cdc.gov/dengue/ for a map of current dengue activity). The former complex classification system has been replaced with 'dengue' or 'severe dengue', and the illness is divided into a febrile phase, critical phase, and recovery phase. Care for dengue is supportive. (See 🕮 Viral haemorrhagic fevers, p. 372)

The first five minutes

ABC. VS, Supplemental O_2 for signs of respiratory distress. Fluid bolus for narrow pulse pressure (< 20 mmHg) even if SBP is normal. Once SBP is low, shock is often irreversible.

History and physical

Dengue ranges from asymptomatic infection, to febrile illness with severe headache and myalgias, to severe haemorrhagic fever with shock. Symptoms usually last for 2–7 days, after an incubation period of 4–10 days from exposure.

Key historical features

- Residence or travel in endemic area
- Risk factors for severe disease: younger age, female, high BMI, history of previous dengue infection
- Timing of symptoms and progression (see phases below)

Signs and symptoms

- Severe retro-orbital pain and headache

• Nausea and vomiting
• Severe myalgias (‘Break Bone Fever’)
• Diffuse lymphadenopathy
• Rash (macules, petechiae, bruising)
• Mild hepatomegaly

Severe disease may begin 3–7 days after first symptoms in conjunction with resolution of fever. Capillary leak syndrome with increased vascular permeability can lead to shock. Warning signs of severe disease include:
• General: AMS; narrow pulse pressure
• ENT: bleeding from gums or nose
• Respiratory: tachypnoea, pleural effusions
• GI: haematemesis, intractable vomiting, severe abdominal pain, ascites

Differential diagnosis

Bacterial sepsis, malaria, typhoid, leptospirosis, influenza, other hemorrhagic fevers, Rickettsial infections (see 🕮 Tick-borne illnesses, p. 376).

Investigations

Usually a clinical diagnosis as definitive testing unavailable in most settings.
• Febrile phase: CBC (mild to moderate leukopenia and thrombocytopaenia occurs in most patients), elevated liver enzymes ◇
• Critical phase: CBC (→HGB due to haemoconcentration, worsening thrombocytopaenia) ◇; ↓ serum protein levels ◈
• Tests for viral nucleic acids or antibodies to viral proteins may be available in select high resource settings ◈

Management

The goal of acute management is to treat pain and manage haemodynamic and haemorrhagic complications to ensure adequate perfusion until resolution.

Supportive care

• Antipyretics (paracetamol) for fever during initial phase
• Analgesia for headache
• Critical phase: once pulse pressure < 20 mmHg (expert recommendation) begin isotonic fluid resuscitation. Once systolic pressure drops, shock may be irreversible
  » Goal: provide only enough fluid to maintain organ perfusion (i.e. keep blood pressure in the low normal range)
  » Avoid overly aggressive fluid resuscitation given capillary leak syndrome
  » Only provide blood for low haemoglobin in the setting of significant ongoing bleeding ◇
  » Only provide platelet transfusions if patient has clinically significant bleeding that cannot be controlled otherwise. Never give platelets solely based on a low platelet count ◇

Critical documentation

• Any history of previous dengue exposure
• Blood pressure (including pulse pressure)
• Location of any bleeding

Disposition

Admit patients with any sign of severe dengue to ICU. Patients with uncomplicated dengue can be managed as outpatients provided they can return for repeat evaluation in 24 hours, and are able return quickly to the hospital for warning signs. Admit all other patients for observation.

147 Tick borne illnesses

Ticks are ectoparasites that live in woody areas and bushes, and tick-borne illnesses are common worldwide. Infections are passed from one tick host to another, including humans, during the tick's blood meal. Many different ticks serve as vectors for human diseases caused by a range of bacteria, virus, protozoa and toxins.

The first five minutes

Assess ABCs, manage respiratory compromise if present. Early IVF for shock.

History and physical examination

Clinical syndromes

- Spotted fevers: (SF: febrile illness with diffuse rash or with one or more eschars.) African tick bite fever (ATBF) is a SF characterised by diffuse rash in addition to eschars. Caused by *Rickettsia* sp.
- Tick Borne Relapsing Fever (TBRF): relapsing fevers lasting a few days (that do not respond to malaria treatment) separated by 1–2 week afebrile periods. Caused by Borrelia sp., spirochetes
- Tick paralysis: paralysis beginning in the legs and ascending to upper extremities. Toxin mediated disease that can be cured by removing the tick (more common in children)
- Haemorrhagic: Crimean Congo Haemorrhagic Fever (CCHF). Viral haemorrhagic fever transmitted by ticks (📖 p. 372)
- Lyme disease: erythema migrans (a single uniformly erythematous oval or circular rash about 16 cm in diameter usually in a 'Bull's eye' pattern)

Key historical features

- History of tick bite or exposure to grasslands and wooded areas
- Presence of eschar
- Relapsing fevers and precise timing
- Flu-like illness: fever, headache, malaise, nausea, vomiting

Differential diagnosis

- Spotted fevers: viral illnesses, dengue fever
- Relapsing fevers: malaria, typhoid, leptospirosis
- Tick paralysis: GBS, transverse myelitis, epidural abscess
- CCHF: other haemorrhagic fevers, severe malaria

Diagnosis

Diagnosis in most African settings will be made on clinical grounds described above.

- Spotted fevers:serology (ELISA, PCR) or tissue biopsy ◈
- TBRF: spirochetes best visualised by dark field microscopy of peripheral blood, bone marrow or CSF. Also by Wright-Giemsa or acridine orange stains
- Tick paralysis: identification and removal of tick is diagnostic
- CCHF: ELISA, real time PCR (📖 p. 372)
- Lyme disease: can do ELISA ◈

Management

The goal of acute anagement is early antibiotic treatment, and supportive care.

Initiate treatment based on high clinical suspicion, do not wait for test results. Though doxycycline usually contraindicated in children, the risk of teeth staining is low, and ill children with suspected tick-borne disease should be treated with doxycycline when no equivalent alternate agent.

- Spotted fevers and African tick bite fever:
  - Doxycycline 100 mg (2.2 mg/kg in children < 45 kg) PO BID × 7 days

• TBRF: tetracycline (500 mg or 12.5 mg/kg PO Q6h) or doxycycline (100 mg PO BID), both × 10d. Second-line erythromycin 500 mg (or 12.5 mg/kg) Q6h × 10d. IV ceftriaxone 2 g QD × 10–14d for CNS involvement. Add antipyretics and cooling strategies as needed
• CCHF: supportive therapy
• Lyme disease: very rare in Africa. Multiple agents can be used. See CDC website for current treatment recommendations

Epidemiology

Table 147.1 Epidemiology of some tick borne illnesses worldwide

Illness	Tick vector	Pathogen	Pathogen	Region	Incubation period
Borelliosis	Blacklegged or deer tick (*Ixodes scapularis*)	Bacteria	*Borrelia burgdorferi* (causes Lyme disease) *Borrelia afzelii* and *Borrelia garinii*	North America Europe	1–2 weeks, but varies Days to months
Rickettsiosis	Ixodes	Bacteria	*Rickettsia, Ehrlichia,* spotted fevers and anaplasma	Worldwide	5–14 days
Babesiosis	*Ixodes scapularis* ticks	Protozoa	*Babesia microti* *Babesia duncani* *Babesia divergens*	USA Europe	1–8 weeks
TBRF	Lone star tick	Bacteria	*Borellia*	Worldwide	1 week
CCHF	*Haemaphysalis* and *Hyalomma* tick	Viral	*Bunyaviridae* family of RNA viruses	East and West Africa, Eastern Europe, Central Asia	1–3 days

148 Cellulitis

Cellulitis, a bacterial infection of the dermis and subcutaneous tissue, is a common presentation, usually associated with *Staphylococcus aureus* and *Streptococcus pyogenes* and minor skin trauma. Immunocompromised states (diabetes, alcoholism, HIV) increase risk and are associated with a wider range of pathogens.

The first five minutes

• ABC, VS, IV if systemically ill
• Pain out of proportion to clinical findings, systemic toxicity, or history of rapidly progressive course over hours suggests aggressive necrotising infection – consult surgical service immediately (do not delay for test results)

History and physical examination

Key historical features

• Onset and duration
• Fever, nausea or other symptoms of systemic infection
• Recent trauma (including human, animal, or insect bites) or invasive procedures (such as surgery, injections)
• Underlying immunosuppressive conditions (diabetes, malignancy, HIV, alcoholism, chronic steroid use, elderly)

Signs and symptoms

• Fever: localised erythema, swelling, warmth and tenderness; regional LAN (assess for lymphangitis)
• Examine (e.g. between toes) for fungal infections or other entry wounds
• Test of range of motion if rash is over a joint
• Facial cellulitis: associated with major complications (e.g. cavernous sinus thrombosis, meningitis). Examine for

orbital involvement and for underlying source, such as dental and sinus infections
- Skin abscesses present with a fluctuant mass of pus surrounded by erythema
- Crepitus indicates gas under tissues, suggesting necrotising infection, and is a surgical emergency
- Mark boundaries of erythema with ink pen and note time drawn on skin to allow monitoring of spread. Progression over minutes to hours indicates life-threatening cause and need for emergent surgical debridement

Possible causes and differential diagnosis

Cutaneous abscess, necrotising fasciitis, lymphangitis, impetigo, localised chemical induced skin irritation. Consider underlying septic arthritis in all cellulitis over a joint.

Investigations

Diagnosis is clinical. Labs may refine the differential:
- XR can diagnose gas-forming infections (low sensitivity) ◇
- US can identify abscess and foreign bodies ◇
- Joint aspiration (NEVER tap a joint through cellulitis) if signs of septic arthritis

Management

The goal of acute management is to treat local infection and to identify underlying causes (e.g. foreign body, septic joint) and systemic involvement. General management includes treatment of pain and fever, elevation of the affected area, and antibiotics.

Antibiotics

See 🕮 Antibiotics p. 968.

Special circumstances

- Treat with IV antibiotics for fever or toxic appearance
- Abscess will NOT resolve with antibiotics alone and must undergo incision and drainage (See 🕮 p. 828)
- Address underlying cause (e.g. remove of FBs, parasites; treat dermatitis)
- Diabetic-related foot cellulitis requires broad coverage for anaerobes and gram negative infections (ampicillin-sulbactam or ceftriaxone)
- Dog and cat bites can be treated with amoxicillin-clavulanate or, if severe, IV ampicillin-sulbactam. Penicillin-allergic patients can be treated with clindamycin and ciprofloxacin (dog bite) or doxycycline (cat bite)
- Treat facial and orbital cellulitis aggressively with IV antibiotics (ampicillin-sulbactam OR clindamycin)

Critical documentation

VS; location, extent and progression of skin findings; presence or absence of associated adenopathy; signs of systemic toxicity.

Disposition

Admit immunocompromised patients and those with evidence of systemic inflammation. Discharge afebrile patients with uncomplicated cellulitis.

149 Toxic shock syndrome

TSS is a term used for several toxin-mediated syndromes related to staphylococcal or streptococcal infection. Bacterial exotoxins act as *superantigens* and cause the release of large quantities of inflammatory cytokines, leading to multisystem failure. Mortality rate is 10% for Staph TSS and 35% for Strep TSS.
- *S. aureus*:
  - Toxic shock syndrome toxin-1, enterotoxin B or C
  - Associated with tampon use; non-menstrual causes account for 50%

- Group A *Streptococcus* (GAS):
  - » Toxic Shock-Like Syndrome (TSLS) or Streptococcal Toxic Shock Syndrome (STSS)
  - » In setting of underlying skin infection/breakdown

The first five minutes

- ABC, VS, facemask O_2, cardiac monitor
- Place two large bore IV lines, draw blood for investigation and start 2L IV crystalloid bolus
- IV antibiotics

History and physical examination

Key historical features

- Tampon use (or other retained foreign body), recent wounds
- Flu like prodromal symptoms
- Timing of skin rash or desquamation

Signs and symptoms

- Flu like prodrome up to two days
- Sepsis; multi-organ dysfunction
- STSS: pain at site of wound; more likely to be abrupt in onset

Differential diagnosis

Leptospirosis, meningococcemia, typhus, acute rheumatic fever, pyelonephritis, osteomyelitis.

Investigations

Staph TSS

- Five clinical criteria: fever (T ≥ 38.9 °C), diffuse macular erythroderma, desquamation (1–2 weeks after rash onset), hypotension and multisystem involvement
- Lab criteria: negative blood or CSF cultures, negative serology for leptospirosis or measles ⟐
- Probable case: meets the lab criteria and four clinical criteria
- Confirmed case: meets the lab criteria and all five clinical criteria

Strep TSS criteria

- Clinical: hypotension plus two or more of: renal impairment, coagulopathy, liver involvement, Acute Respiratory Distress Syndrome, erythematous macular rash, soft tissue necrosis
- Lab: isolation of GAS ◇
- Probable case: isolation of GAS from a non-sterile site (throat, vagina, sputum)
- Confirmed case: isolation of GAS from a normally sterile site (blood, CSF or, joint, pleural, or pericardial fluid)

Management

The goal of acute management is rapid antibiotic administration and supportive care. Supportive care: IV fluid resuscitation; vasopressors ◇; mechanical ventilation as necessary ⟐.

Other acute care

- Identify (and debride if possible) focus of the infection ◇
- **Empiric parenteral antibiotics** (see Table 149.1)
- **Cover both staph and strep if source unknown**

- IV immunoglobulin (400 mg/kg IV over six hours) if no clinical response within six hours of aggressive resuscitation ⟐

Critical documentation

Serial VS and response to interventions, suspected source and removal/debridement.

Disposition

Admit or transfer all patients for ICU care when available.

Table 149.1 Antibiotics for TSS

	Option 1	Option 2 (Pen Allergic)	Comment
GAS	Penicillin G and clindamycin	Clindamycin and macrolide OR fluoroquinolone	Resistance developing to macrolides and quinolones
MLS resistant GAS	Pen G and vancomycin	Vancomycin or linezolid	Generally also resistant to clindamycin
Methicillin sensitive *S. aureus*	Clindamycin AND cloxacillin OR nafcillin OR cefazolin	Clarithromycin and clindamycin	
Methicillin resistant *S. aureus*	Vancomycin AND clindamycin or linezolid	Rifampicin AND linezolid	
Glycopeptide resistant *S. aureus*	Linezolid AND clindamycin	Daptomycin	Increasing overall, but prevalence highly geographically variable
MLS = Macrolide, Lincosamide, and Streptogramin B			

Adapted from Lappin E & Ferguson AJ. Gram positive toxic hock syndromes. Lancet Infect Dis. 2009;9(5):281–90.

150 Post-infectious conditions

Infections may trigger autoimmune or inflammatory reactions in which symptoms or organ damage develop after the infectious agent has been cleared. These conditions are difficult to diagnose, but important to consider, as they are often on the DOX for acute infections.

Syndromes

Haemolytic Uraemic Syndrome (HUS)

- Three features: microangiopathic haemolytic anaemia, thrombocytopenia, acute kidney injury
- Associated with *E. coli* produced shiga-toxin, or causing a diarrhoeal illness or dysentery (70%). However, shiga-toxin-producing *E. coli* that cause urinary tract infection has been reported in cases of HUS. Shigella infection itself can also induce HUS; this form caries higher mortality and greater risk of permanent renal dysfunction

Clinical features

- Most children have prodrome consistent with gastroenteritis
- Neurologic (seizures, strokes); cardiac (pump dysfunction)
- GI (bowel necrosis, perforation, haemorrhagic colitis)
- Renal (range from proteinuria/haematuria to oligouric renal failure requiring dialysis)

Diagnosis

- Clinical; CBC (anaemia and thrombocytopaenia), and evaluation of renal function (ideally measurement of glomerular filtration rate; alternately urine dipstick and microscopy). Coagulation studies should be normal.

Management

- Do not administer antibiotics for *E. coli* infection

- Use standard medication for hypertension
- Cautious hydration, monitor output and clinical status to avoid fluid overload
- Transfuse packed RBCs for severe symptomatic anaemia ◇
- Avoid giving platelets unless life-threatening bleeding present ◈
- Treat hyperkalaemia as needed ◇; dialysis for renal failure occurs ◈

Post-infectious acquired neutropaenia

- Acquired neutropaenia can develop after infection (bacterial sepsis, parvovirus, influenza, measles, cytomegalovirus, EBV, HIV, TB, malaria)
- Characterised by decline in absolute neutrophil count (ANC) below 1 500/mm^3
  » ANC = WBC × (% bands + % neutrophils)
  » Mild = 1 000–1 500/mm^3; moderate = 500–1 000/mm^3, severe = < 500/mm^3
- As ANC drops < 500/mm^3, risk of serious bacterial or fungal infection increases

Clinical features

- Infection signs, sore mouth, low-grade fever, weakness, malaise, dyspnoea
- In severe cases, hypotension and septic shock

Diagnosis

- Clinical; post infection timing. CBC with differential to make diagnosis ◇

Management

- Supportive care, limit contact, strict hand washing for providers
- Place in positive pressure isolation ◈
- Antibiotics for ANC < 500 and fever: 3rd gen cephalosporin as single agent; add aminoglycoside and antifungal for worsening/no response after four days ◇

Post-streptococcal glomerulonephritis

- Development of oedema, haematuria (tea-coloured urine), proteinuria, hypertension, 7–21 days after group A beta-haemolytic strep infection to skin or throat
- Typically in children 2 months–2 years of age
- Glomerular injury has already occurred by time of presentation

Clinical features

- Varying degrees of hypertension, oedema and haematuria.

Diagnosis

- Clinical picture and blood chemistry show elevated K, BUN and creatinine, urine proteinuria ◇; extent of renal dysfunction correlates with severity
- Total haemolytic complement and C3 levels ◈ decreased in most cases
- Positive throat culture ◇ or elevated strep antigen titre ◈ highly suggestive

Management

- Supportive care
- Treat severe hypertension with labetalol and admit. Nitroprusside or diazoxide can be used in refractory cases. Furosemide if needed ◇
- Consult nephrologist ◈ for: hypotension, oedema, recurrent haematuria, oliguria, nephrotic range proteinuria, severe azotaemia
- Long term out-patient follow-up may be necessary to manage hypertension

Guillain-Barré syndrome

• Immune-mediated acute demyelinating disease affecting peripheral nerves
• Develops two weeks after gastrointestinal or upper respiratory infection
• Associated with campylobacter, CMV, EBV, mycoplasma pneumonia

Clinical features

• Initially peripheral paraesthesia, then symmetrical proximal muscle weakness of the lower extremities, then progresses to involve arms and trunk
• Can progress to respiratory failure due to weakness of respiratory muscles. Autonomic dysfunction (labile BP) can also be seen. Symptoms typically peak in 2–4 weeks, followed by a plateau phase, and slow recovery over 2–4 weeks

Diagnosis

• Clinical diagnosis; labs to exclude other disease entities

Management

• Due to potential for respiratory failure, admit until in plateau phase. Transfer to centre with ICU and capacity for prolonged respiratory support ⟐
• IVIG or plasmapheresis to reduce the duration of symptoms ⟐

Reactive arthritis (formerly Reiter's syndrome)

• Autoimmune, may be isolated oligoarthritis or triad of arthritis, nongonococcal urethritis, and conjunctivitis
• Develops 1–4 weeks after gastrointestinal or genitourinary infection
• Associated with *Shigella*, *Salmonella*, *Campylobacter*, *E. coli*, and *Chlamydia*

Clinical features

• General malaise, fever, fatigue, oligoarthritis of weight-bearing joints
• Eye: conjunctivitis or uveitis
• Genital: discharge, tender prostate, vulvovaginitis
• Mucocutaneous: painless vesicular oral lesions, keratoderma (yellow to brown papules that desquamate, often on soles of feet), erosion of glans penis

Diagnosis

• Clinical diagnosis; test to rule out septic arthritis, other infectious conditions
• Slit lamp exam to identify uveitis ⟐; elevated inflammatory markers ◇

Management

• Screen and treat for STI and HIV per local guidelines
• Rest and NSAIDs for arthritis; consider local corticosteroid injections or oral for refractory cases
• For uveitis, consult ophthalmology ⟐, drops or oral corticosteroids

Reye syndrome

• Form of acute toxic-metabolic encephalopathy; occurs most commonly in children after viral illness (e.g. chicken pox or influenza). Affects brain and liver
• Aspirin use during the viral illness is a major risk factor
• Fatty infiltration of the liver leads to hypoglycaemia, and hyperammonemia causing cerebral oedema, and increased ICP

Clinical features

- Occurs during recovery from viral syndrome, and presents with vomiting, AMS, and high fever, progressing to seizures and coma, and may have a rash on palms and soles. Hepatomegaly is common
- Diagnosis is clinical, though labs show elevated LFT ◇, hyperammonemia ◈, hypoglycaemia, and metabolic acidosis

Management

- Primarily supportive; monitor blood glucose and administer dextrose infusion as needed for hypoglycaemia, elevate head of the bed (30^0), avoid excessive hydration
- Furosemide for volume overload ◇; phenytoin or benzodiazepines for seizures ◇
- Intubation for severe cases (to maintain PCO_2 in normal range) ◇
- Mannitol for coma ◇

Paediatric Autoimmune Neuropsychiatric Disorder associated with Group A Streptococci infection (PANDAS)

- A form of obsessive compulsive disorder (OCD) and/or a tic disorder (Tourette Disorder) associated with Group A strep infection

Clinical features

- Presents with onset or increase in OCD symptoms and motor hyperactivity
- Abrupt onset, episodic, sometimes improves with treatment of strep infection

Diagnosis

- Clinical presentation in the setting of recent infection

Management

- Treat underlying strep infection if present. Treat OCD and tic symptoms with standard medications (SSRIs and dopamine blocking agents, respectively).

Acute Disseminated Encephalomyelitis (ADEM)

- Inflammatory auto-immune CNS demyelinating disease associated with preceding infection or vaccination
- Typically involves descending motor tracts, optic nerves and spinal cord

Clinical features

- Typically affects prepubertal children
- History of infectious illness or vaccination 1–2 weeks prior to onset
- Febrile prodrome, headache, vomiting, confusion, abrupt onset of AMS
- Limb ataxia, paresis, loss of voluntary motor control
- Can present with bilateral loss of vision, DIB, bladder dysfunction

Diagnosis

- Clinical diagnosis; CSF (elevated myelin basic protein), CT or MRI (multiple inflammatory lesions) ◈

Management

- High dose corticosteroids ◇; IVIG or plasmapheresis if no response to steroids ◈

151 Needle-stick injury

Workplace needle-stick injuries carry variable risk for transmission of blood-borne diseases (HIV, Hepatitis B (HBV) and Hepatitis C (HCV)). Transmission risk relates to exposure type, local prevalence, and source viral load.

Overall transmission risk is 6–30% for HBV, 0–7% for HCV, and 0.3% for HIV.

The first five minutes

Irrigate with copious water and mild disinfectant (chlorhexidine gluconate) or hand-cleaning solution. Avoid bleach or iodine. Do not squeeze/rub wound.

History and physical examination

Key historical features

- PMHx of exposed provider; HIV, HBV and HCV status**;** immunisation history
- Risk assessment:
  - » HIV status of source; viral load and medication regimen if positive
  - » **High risk features:** hollow needle, presence of visible blood, needle from patient's artery/vein, and deeper stick

Investigations

- With consent, test source and exposed person for HIV
- Consider the 'window' period during which new HIV infection may only be detectable by antigen-based (rather than antibody-based) testing ◈
- Test source **and** exposed person for HBV and HCV ◇

Management

The goal of acute management is risk stratification and timely prophylaxis to prevent infection.

Post exposure prophylaxis (PEP)

- HBV: assess pre-exposure vaccination status
- HCV: none
- HIV: immediate antiretroviral therapy (ART), even if HIV testing unavailable
  - » Ideally start within two hours of exposure
  - » Refer to national protocols
  - » Pregnancy NOT a contraindication, though avoid tenofovir + emtricitabine
- Update tetanus prn

Counselling

- Counsel exposed patient: adherence and side effects of PEP. Protect sexual partners with condoms until repeat testing complete at 6 months
- Exposure-related mental health impact. Social support and safety

HIV post-exposure prophylaxis with ART

Indications

Exposure within 72 hours (ideally start within 2 hours)
Source is HIV+ or HIV-unknown
Exposed person is believed HIV-negative

Contraindications

Exposure > 72 hours prior
Exposure without risk of transmission
Exposed person is HIV+

WHO PEP recommendations

Two drug regimen

Indications:

Source HIV-unknown with local ART resistance of < 15%

Source is HIV+ with low suspicion for resistance

Zidovudine* + lamivudine

*Alternative regimens (**Emtricitabine may replace lamivudine:)

Tenofovir + lamivudine**

Stavudine +lamivudine**

Three drug regimen

Source is HIV-unknown and local ART resistance is > 15%

Source is HIV+ with known resistance

Zidovudine* + lamivudine plus lopinavir/ritonavir

Side effects and monitoring

Common minor side effects: nausea, fatigue, headache, vomiting, diarrhoea Severe side effects include: nephrolithiasis, hepatitis, pancytopaenia, other Monitor for drug toxicity, with laboratory testing as clinically indicated Monitor HGB with zidovudine

Arrange follow-up

Complete 28 day regimen of ART. If source is HIV-negative and outside window period, discontinue ART. Repeat HBV, HCV, HIV testing six months post-exposure.

Critical documentation

Details of exposure, presence of high risk factors; test results of source patient.

Disposition

Discharge with follow-up.

152 Cerebral venous thrombosis

Cerebral venous thrombosis (CVT) is a rare but life-threatening condition. Aseptic CVT results from trauma, iatrogenic injury, or pro-thrombotic states. Septic CVT involves a thrombus with associated bacterial superinfection, usually as a complication of sinus, ear, tooth, nose or mouth infection. Cavernous sinus thrombosis is the most well-known form, but CVT occurs in many locations. Signs and symptoms are related to cerebral parenchymal lesions local to thrombus (e.g. haemorrhage, oedema), or decreased CSF absorption leading to elevated ICP.

The first five minutes

- ABC, VS, facemask O_2, IV; cardiac monitor. For sepsis, 2 large bore IV and IVF
- 12 lead ECG

History and physical examination

Key historical features

Ask about history of trauma; recent ENT, dental or neurosurgical procedures; diabetes or immunocompromised states (HIV, malignancy, steroid use).

Signs and symptoms

General: headache, fever, lethargy, papilledema, vision problems.
Associated with specific vascular locations:
- Cavernous sinus: ocular signs (red or painful eye, proptosis; III, IV, V1, V2 cranial nerve palsies. decreased corneal reflex, decreased visual acuity)
- Sagittal sinus: bilateral deficits, seizures
- Cortical vein: sensorimotor deficits and seizures
- Isolated lateral sinus: may be isolated headache and/or intracranial hypertension
- Jugular vein, lateral sinus or posterior fossa veins: tinnitus, multiple cranial nerve palsies
- Straight sinus and branches (deep system): coma, AMS, motor deficits

Possible causes and differential diagnosis

- Aseptic: inflammatory and autoimmune diseases, including malignancy, trauma, coagulopathic states, post-surgical inflammation
- Septic: infection spreading from ENT, dental, or CNS. *Staphylococcus aureus* most common, then *Streptococcus* species. Less common organisms include gram-negative bacilli, anaerobes, fungi (*Aspergillus*, *Rhizopus*)
- Differential diagnosis: other ischaemic or haemorrhagic stroke; cerebral neoplasm; other cerebral inflammatory diseases; other infectious (orbital cellulitis, preseptal cellulitis, brain abscess, meningitis); other causes of seizure and weakness (see 🕮 Infectious diseases, p. 310–386)

Investigations

Labs: CBC, blood cultures, CSF studies ◇; coagulation studies, D-dimer, ◈
Imaging:
- CT brain with contrast: normal in 30%; direct evidence of clot (hyperdensity or filling defect) in 30%; indirect evidence (haemorrhagic lesions, oedema in non-arterial distribution) common ◈
- CT venography shows flow/filling defects and has very high sensitivity and specificity ◈
- MRI with venography: most sensitive, study of choice ◈

Management

The goal of acute management is to manage elevated ICP, identify and treat infection, and prevent clot propagation. Consult neurology when available; haematology for thrombotic work-up; if septic thrombus, consult neurosurgery for possible drainage and ENT or oral surgery as needed for source.
- Anticoagulation ◈: heparin: unfractionated should be initiated immediately, if no contraindications to anticoagulation
- Antibiotics: initiate broad-spectrum empiric antibiotics with good CNS penetration and coverage for *S. aureus* (cloxacillin, clindamycin, vancomycin (if MRSA)), strep (ampicillin, clindamycin, ceftriaxone, vancomycin), and gram negative bacteria (ceftriaxone, gentamicin, chloramphenicol)
- Associated conditions: patients with CVT may be severely ill and require emergent management of elevated ICP, other clot (pulmonary embolism) if hypercoaguable, and sepsis

Critical documentation

Serial VS and mental status exam; surgical consultations; fluids, anti-coagulation and antibiotics; all diagnostic imaging results.

Disposition

Admit all patients with CVT to ICU care as soon as possible.

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Part

4

I. Metabolic and nutritional

153 Approach to the child with severe acute malnutrition

Malnutrition in children can present as a complex medical emergency (termed severe acute malnutrition – SAM) and may include shock, hypothermia, hypoglycaemia, infection, metabolic and electrolyte derangements and immune dysfunction.

Malnutrition states are a spectrum of disease including marasmus (deficiency of caloric intake typified by wasting) and kwashiorkor (deficiency of protein intake typified by wasting and oedema).

Definition

SAM is defined as the presence of severe visible wasting (< 70% weight-for-height or < -3 SD) and/or a mid-upper arm circumference measurement of < 11.5 cm and /or symmetrical oedema involving at least the feet in a child aged 6 months to 5 years.

The WHO Emergency Triage, Assessment and Treatment (ETAT) describes two very simple checks to identify SAM:

1. Severe visible wasting – particularly face, limbs, axillary area and buttocks
2. Bilateral pitting oedema of feet – often associated with puffy face and eyes, skin sores, nappy rash and depigmentation of hair

All children with SAM should be admitted for in-patient management. The appropriate level of in-patient facility depends on factors such as age, appetite and complications (e.g. heart failure, fever, sepsis) (Figure 153.1).

To assess the severity of metabolic derangement in the child with SAM, an appetite test is recommended (Figure 153.2). A child with poor appetite is considered at high risk and requires careful stabilisation and specialised care. WHO recommends a 10-step approach to the management of SAM:

Resuscitate

Step 1

Address circulatory failure/shock. May be due to sepsis, dehydration or dysrhythmia. Resuscitate with caution: rapid fluid boluses may precipitate heart failure and worsen outcome. Give 10 ml/kg IV NS or ringers lactate over 30 min; repeat if shock persists. Once shock corrected rehydrate over 24 hours orally or via NGT, avoiding further IVF unless paralytic ileus or excessive vomiting. If able to tolerate NGT or oral feeds, use rehydration solution for malnutrition (ReSoMal) or locally used malnutrition formula: if above unavailable use homemade SSS (sugar salt solution: 1 L water, ½ tsp salt, 8 teaspoons sugar, plus oral KCl syrup)

- Give 5 ml/kg every 30 min for 2 h, PO or NGT; then 5–10 ml/kg/h depending on on-going losses and patient request

Table 153.1 WHO ReSoMal low sodium oral rehydration solution

Ingredient	**Quantity**
Water (boiled and cooled)	2 L
WHO-ORS	1 packet (usually for 1 litre)
Sugar	50 g
Electrolyte solution (see Table 153.2)	40 ml

Step 2

Treat and prevent hypoglycaemia.

- Conscious child – oral/NGT 50 ml bolus of 10% glucose or 10% sucrose solution (1 rounded teaspoon of sugar in 3.5 tablespoons water)
- Unconscious child:
  - » 5 ml/kg IV/IO bolus dextrose 10%, followed by maintenance fluids containing 5–10% dextrose plus electrolytes (e.g. ½ Darrow's Dextrose)
  - » If no IV/IO 5 ml/kg NGT bolus dextrose 10% and/or place 1 ml of 50% dextrose in buccal/sublingual space
- Re-check glucose after 15 minutes, repeat bolus (2 ml/kg of 10% dextrose) if needed, and then monitor glucose Q3h and feed initially Q2h

Step 3

Treat and prevent hypothermia. If rectal temp < 35.5°C (or axillary temp < 35°C):

- Feed straight away and then every 2–3 hours
- Place the child on his/her mother's chest
- Wrap the child in warmed clothes and blankets and cap
- Consider a warming light
- Closely monitor temperature
- Warm any oral or IV solutions

Step 4

Treat infection. Consider empiric antibiotics as may not manifest typical signs. Bring vaccinations up to date.

- If no evidence of infection and not severely unwell: co-trimoxazole PO 5 ml BID 5 days (2.5 ml if < 6 kg). (5 ml = 40 mg TMP and 200 mg SMX) or amoxycillin PO 30 mg/kg/T10 × 5 d
- If severely ill or has complications: ampicillin 50 mg/kg IM/IV Q6h × 2 d, then oral amoxicillin 15 mg/kg TID × 5 d AND gentamicin 7.5 mg/kg IM/IV daily × 7 d. If recently in hospital, treat nosocomial infections. Consider treatment for malaria

Address electrolyte and micronutrient deficiencies

Step 5

Correct electrolyte imbalances. May be hypo- or hyper-Na; avoid high sodium solutions. On-going repletion may be necessary if stores are depleted. Recheck levels as needed.

• Extra K 3–4 mmol/kg/d (added to oral fluids as KCl syrup)
• Extra Mg 0.4–0.6 mmol/kg/d (added to oral fluids)
• Phosphate supplementation 1 mmol/kg/d
• Prepare low salt foods

Table 153.2 WHO electrolyte repletion solution

Electrolyte	Quantity (g)
Potassium chloride	224
Tripotassium citrate	81
Magnesium chloride	76
Zinc acetate	8.2
Copper sulfate	1.4
Water: make to	2 500 ml

Step 6

Replace micronutrient deficiencies. WHO recommend against giving iron therapy initially; may increase mortality. Vitamin A PO: > 12 months, 200 000 IU; 6–12 months, 100 000 IU; 0–5 months, 50 000 IU (unless definite evidence that dose has been given in last month).

Supplements PO, daily for at least two weeks:
• Multivitamins drops (without iron) daily
• Folic acid 1 mg/d (5 mg on Day 1)
• Zinc 10 mg daily in children <10 kg, 20 mg daily in children > 20 kg
• Copper 0.3 mg/kg/d
• Iron 3 mg/kg/d (only start when oedema resolving, usually after two weeks). Transfuse if Hb < 4 or respiratory distress from anaemia

Feeding

Step 7

Stabilisation phase: over first week feeding volume may increase and frequency decrease from Q2h to Q4h.
• Small frequent feeds of low osmolarity, low lactose and low sodium, start 80 ml/kg/day in oedematous malnutrition
• If patient unable to take all feeds, do then NGT. Start F-75 milk based formula or similar
• Goal: 100 kcal/kg/day, 1–1.5 g protein/kg/day
• 100–120 ml/kg/day fluid depending on the extent of oedema, building up by 20 ml/kg/day depending on weight gain and severity
• Continue breastfeeding but supplement as necessary
• Monitor closely both quantity ingested and losses

Table 153.3 F-75 Formula Recipe (WHO)

Ingredient	Quantity
Dried skim milk	25 g
Sugar	100 g
Vegetable oil	30 g
Electrolyte/mineral solution	20 ml
Water: make to add up to:	1 000 ml

Step 8

Rehabilitation phase: transition when appetite returned.

Goal is to achieve more rapid weight gain (> 10 g gain/kg/day):
• Milk-based F-100 Catch-Up (100 kcal/100 ml and 2.9 g protein/100 ml) or similar local foods with comparable

protein and caloric content may be used

- Replace F-75 feeds with an equal volume of F-100; increase F-100 by 10 ml steps until some remains uneaten
- Monitor closely for evidence of heart failure
- Feed at least every four hours with now unlimited amounts
- Goal: 150–220 kcal/kg/d, 4–6 g protein/kg/d

Table 153.4 F-100 Formula (WHO)

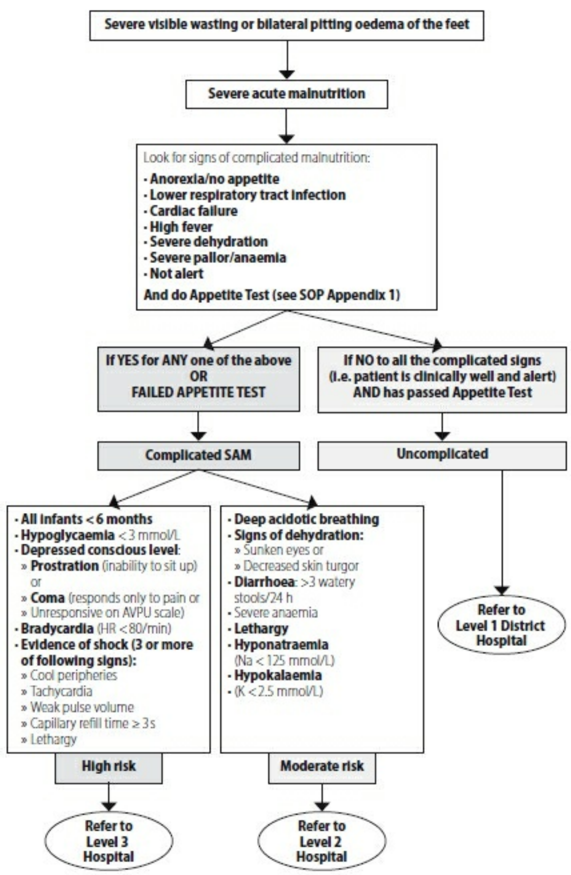
Ingredient	Quantity
Dried skim milk	80 g
Sugar	50 g
Vegetable oil	60 g
Electrolyte solution	20 ml
Water: make to add up to:	1 000 ml

Step 9

Emotional support. Provide a loving, caring environment with consideration for physical activity (as is possible), family involvement, and play time.

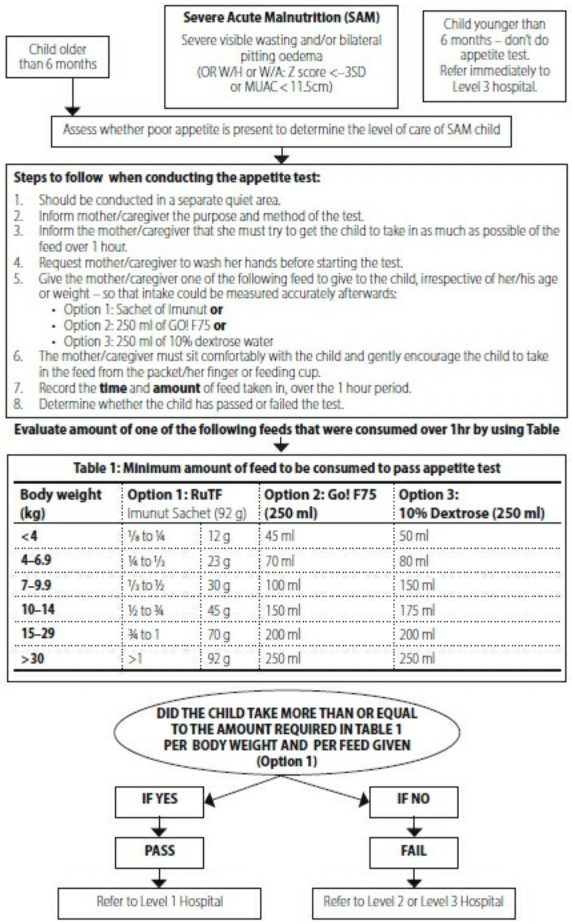
Step 10

Recovery and follow-up. A child who has attained 90% of weight for length has recovered.



Source: University of Cape Town, Emergency Medicine Guidelines, 2013.

Figure 153.1 Classification and referral of severe acute malnutrition



Source: University of Cape Town, Emergency Medicine Guidelines, 2013.

Figure 153.2 Appetite test algorithm

154 Approach to hyperglycaemia

Hyperglycaemia is common among patients presenting acutely, and may signal a broad range of disease processes. A random (non-fasting) plasma glucose > 11.1 mmol/l suggests diabetes mellitus; hyperglycaemia may also be a response to other acute conditions such as stroke or MI.

The first five minutes

• ABC, VS, IV, glucose level

History and physical examination

Key historical features

Medical history (e.g. diabetes, chronic renal disease) and medication, recent oral intake, and medication compliance. Chest pain, shortness of breath, neurologic dysfunction (visual, motor, sensory, or speech disturbance; confusion), fever, and vomiting/diarrhoea.

Signs and symptoms

Hyperglycaemic patients may be completely asymptomatic. May have tachypnoea, tachycardia, hemiplegia/hemiparesis, aphasia/dysphasia, diaphoresis, AMS, seizure or post-ictal state, and polydipsia or polyuria.

Possible causes and differential diagnosis

The differential diagnosis of hyperglycaemia is extremely broad, and includes:

- Diabetic crisis (e.g. HHS or DKA)
- Endocrine disorder: pituitary/hypothalamic dysfunction, hyperthyroidism
- Myocardial infarction
- Stroke
- Exacerbation of pulmonary disease (e.g. asthma or COPD)
- Pancreatic neoplasm
- Infection
- Seizure
- Medications (e.g. corticosteroids, non-compliance with diabetic regimen)

Investigations

Initial investigations should focus on identifying potentially life-threatening conditions, and assessing major organ function. Patients with asymptomatic, mild hyperglycaemia require little or no testing.

Management

The goal of acute management is detection and treatment of acute, life threatening complications, and moderation of blood glucose concentration. Therapy depends upon aetiology, severity, and associated symptoms or conditions.

- Treat underlying causes as per specific conditions
- In uncomplicated or mild cases, IV hydration alone may be adequate to re-establish euglycaemia. A single dose of subcutaneous regular insulin may also be given to patients with a known history of diabetes mellitus, with a target glucose level of 8.3 mmol/l. Caution if renal insufficiency

Critical documentation

Blood glucose level, suspected cause, investigation findings and treatment administered.

Disposition

Admit patients as per the underlying condition.

- In the case of new-onset type 2 diabetes mellitus, consider starting on a low-dose oral agent, but renal function MUST be checked first. Metformin is contraindicated in patients with serum creatinine > 130. Ensure that the patient receives appropriate diabetes education prior to discharge, and close outpatient follow-up
- If the patient is a known diabetic, hyperglycaemia may represent a problem with dietary or medication compliance, a change in his/her response to the existing medication regimen, or a worsening of the disease process. Follow up in primary care within seven days

155 Hypoglycaemia

Low blood glucose is a common cause of AMS, and can mimic almost any neurological presentation. While typically correctable, if unaddressed it may progress to coma and death. Both the symptoms and the blood glucose level at which they occur is variable (especially in diabetics), but a glucose < 4 mmol/l (70 mg/dl) in adults and < 3.5 mmol/l in children is considered low. In non-diabetic children it is a sign of serious illness.

The first five minutes

- ABC, VS, IV access, glucose level
- Administer glucose (if no IV, 1–2 ml 50% dextrose or sugar to buccal mucosa)

History and physical examination

Key historical features

History is often only possible after treatment. Ask about medication history and recent food intake. Beta-blockers may impair or blunt initial symptoms. In diabetics, ask about any dose changes.

Signs and symptoms

Findings are usually non-specific, and include AMS, tachycardia, diaphoresis, hypothermia, loss of coordination, Focal neurologic signs including stroke syndrome, tremor or seizure activity.

Possible causes and differential diagnosis

The differential is broad and includes most causes of neurologic dysfunction and AMS.

Causes include (multiple causes may co-exist):

- Poor nutritional intake
- Medication effect (insulin or oral anti-diabetic agents)
- Intoxication (alcohol, cocaine, amphetamine)
- Hepatic disease (impaired gluconeogenesis)
- Infection
- Renal disease (may ↑ effect of medication)
- Insulin secreting tumours

Investigations

Beyond the initial rapid glucose, investigations are directed at the suspected cause. Diabetics with simple medication-related hypoglycaemia may not need labs but always consider new renal insufficiency.

Management

The goal of acute management is restoration of blood glucose concentration.

- If the patient able, give food/drink
- Otherwise, give IV Dextrose (infants and children: 5 ml/kg of 10% dextrose, commence dextrose-containing maintenance; adults: 25–50 ml 50% or 125–250 ml 10%, continue 5%–10% dextrose maintenance), re-check in 15 min ◇
- If no IV access, give 10% sucrose solution (1 rounded teaspoon of sugar in 3.5 tablespoons water) orally or via NGT, oral Glucogel above dose of dextrose via NGT ◇ and/or place 2–5 ml of 50% dextrose in buccal space ◇
- For persistent hypoglycaemia, continuous IV dextrose infusions (5%–10%) in children the fluids must have added electrolytes (e.g. ½ DD, NOT plain D5–10%) ◇. 🕮 Volume resuscitation paediatrics p. 27

Critical documentation

Document symptoms, serial blood glucose levels, aetiology, and response to treatment.

Disposition

Admit all children who have been hypoglycaemic as it is a sign of serious illness. Admit all patients who are hypoglycaemic due to longer acting insulins or oral anti-diabetic medications. There is a high 24–48 hour risk of recurrent hyperglycaemia with oral agent overdose, especially with sulphonylureas. (🕮 Oral anti-diabetic agent p. 682).

156 Hyperglycaemic hyperosmolar state

Like DKA, hyperglycaemic hyperosmolar state (HHS) is potentially life-threatening and results from a relative or absolute insulin deficiency. HHS is defined by AMS, hyperglycaemia and hyperosmolarity, leading to profound dehydration with little or no ketoacidosis. In HHS, plasma glucose is often much higher than in DKA, often exceeding 33 mmol/l. Patients may present with neurologic deficits resembling seizure or stroke. The most common precipitating factors are inadequate insulin therapy, and infection.

The first five minutes

- ABC, VS, O_2, IVF, glucose level, cardiac monitor
- Secure airway and resuscitate – as needed

History and physical examination

Key historical features

Ask about recent illness, fluid intake, co-morbid conditions (especially renal and cardiac disease) and medication history.

Signs and symptoms

Symptoms may include thirst, polyuria or oliguria, vomiting, weakness/lethargy, and confusion.

Exam findings include AMS (particularly lethargy or coma), poor skin turgor, hypotension, tachycardia, focal seizures, hemiplegia, hemianopsia, or other stroke-like syndromes, and peripheral vasodilation.

Possible causes and differential diagnosis

The differential includes DKA and alcoholic ketoacidosis, trauma, sepsis, intoxication (especially aspirin or paraldehyde), MI, starvation ketosis, stroke, and post-ictal state.

Investigations

- Lab: CBC, electrolytes (adjust sodium for hyperglycaemia (= measured Na 0.024* [serum glucose – 100]); K may be falsely elevated but total body stores will be low; increased serum osmolality correlates directly with impaired mental status), renal, glucose, ABG (usually only mild acidosis), urinalysis, pregnancy test ◇; Mg, PO4 ◈
- ECG ◇

Other investigations should be directed towards finding a cause.

Management

The goal of acute management is correction of acute electrolyte imbalances and intravascular volume depletion.

- Secure airway as needed (avoid succinylcholine, as it may worsen hyperkalaemia)
- IV fluids: NS boluses of 250 ml until well-perfused, then 150–250 ml/hr based upon cardiopulmonary status and osmolarity. Switch to 0.45% NS if corrected sodium is elevated
- Insulin: normal glucose levels may be achieved with IVF alone. If glucose does not drop by 3–4 mmol/l per hour with IVF, consider a 0.1 unit/kg IV insulin bolus (max 10 units). When glucose < 16 mmol/l, switch to 5% dextrose solution if continued insulin use
- Potassium: check K frequently until stabilised. If < 4.5 mmol/l, add 20 mmol/hr to fluids
- Avoid bicarbonate administration unless pH < 6.9
- Treat potential causes (e.g. infection, ACS)

Critical documentation

Document clinical findings, labs, therapy administered and response, and probable cause.

Disposition

Admit all patients, preferably to a high care setting ◈.

157 Diabetic ketoacidosis

Diabetic ketoacidosis (DKA) is caused largely by a relative or absolute lack of insulin, accompanied by dehydration.

Patients in DKA typically exhibit hyperglycaemia, volume depletion and acidosis related to the production of ketones. Inciting factors for DKA include disruption of medication dosing, stress, infection, stroke, trauma or MI.

The first five minutes

- ABC, VS, glucose, IVF – 2 L bolus; cardiac monitor

History and physical examination

Key historical features

Ask about history of diabetes, any previous DKA; typically a 1–3 day history of thirst, polyuria, blurred vision, abdominal pain.

Signs and symptoms

Symptoms are often non-specific and include dizziness, vomiting, abdominal pain, polydipsia and polyuria, headache, weakness and fatigue. On exam, look for tachypnoea (Kussmaul's respiration), hypotension, tachycardia, fruity breath odour (acetone), abdominal tenderness (acidosis or comorbid abdominal pathology), coma, dry mucous membranes and delayed capillary refill. Occult infection is common.

Possible causes and differential diagnosis

Nearly any illness or infection can precipitate DKA. In the differential, consider:

- Other causes of anion gap metabolic acidosis
- Pancreatitis
- Perforated gastric/duodenal ulcer
- Other causes of AMS
- Toxic ingestions
- Hyperglycaemic hyperosmolar state
- Stroke
- MI

Investigations

Investigations should be directed at the search for causes and complications.

- Labs: CBC, electrolytes, renal (a high anion gap is typical), glucose, urinalysis (dipsticks only detect 1 ketone – acetoacetate – which may be absent in DKA), ABG ◇; serum ketones, lactate ⟐
- ECG ◇
- Imaging: CXR

Management

The goal of acute management is restoration of intravascular volume and correction of acidosis.

- IVF 2 l IV bolus rapidly. Fluid deficits may be significant (> 5 l); clinical condition, volume status, response to treatment and elimination of anion gap and acidosis should guide therapy. Fluid replacement will address much of the hyperglycaemia
- Insulin: IV – no bolus is required; give short acting continuous infusion 0.1units/kg/hr. Alternatively, SC boluses (0.1 units/kg SC Q2h) with careful monitoring of glucose level
- Insulin should be continued until the anion gap is closed; use half insulin dose and add dextrose-containing IV solution when blood glucose ≤ 14 mmol/l (250 mg/dl). Don't drop blood glucose faster than 5 mmol (90 mg/dl) per hour
- Paediatrics: in children, fluid resuscitation and insulin therapy must be carefully monitored to prevent cerebral oedema. Initial fluid repletion should be 10–20 ml/kg NS over 1–2 hours (no initial bolus), followed after 1–2 hours with insulin therapy. Total volume administered over the first four hours should not exceed 40–50 ml/kg. SC insulin therapy in children must be carefully monitored as bolus insulin may elicit cerebral oedema. In the

absence of shock or severe, life threatening disease, insulin and IV fluid resuscitation should proceed slowly

- Potassium: although initial lab K may be normal or high, body levels are low and it needs replacing. Once K is known to be < 5.5, add to IVF (e.g. 20 mmol/hr) and regularly monitor blood levels ◇. If K < 3, hold insulin until repleted
- Provide IM or slow IV Mg replacement ◈

Always identify and treat precipitating cause.

Critical documentation

Document history or new diagnosis of diabetes, blood glucose level, lab results including presence of ketones, and treatment administered including response.

Disposition

Admit all patients, preferably to a high care setting. Patients with a new diagnosis of Type 1 diabetes should receive education and training in insulin self-administration.

158 Alcoholic ketoacidosis

Alcoholic ketoacidosis affects those with poor underlying nutrition who ingest large quantities of alcohol. It is characterised by anion gap metabolic acidosis, significant volume depletion, presence of ketones and normal or low blood glucose. Comorbidities include pancreatitis, perforated ulcer, or alcohol withdrawal.

The first five minutes

- ABC, VS, IV, glucose level

History and physical examination

Key historical features

Patients have a history of heavy alcohol abuse but may have limited alcohol and food intake immediately prior to presentation.

Signs and symptoms

Ask about vomiting, anorexia, abdominal pain (due to acidosis or abdominal pathology such as pancreatitis), dizziness, dyspnoea, and confusion or lethargy.

Physical findings generally relate to volume depletion and chronic alcohol use: tachycardia, hypotension, tachypnoea, acetone breath, abdominal tenderness and AMS (confusion to severe obtundation and coma).

Possible causes and differential diagnosis

- Other causes of anion gap metabolic acidosis such as lactic acidosis/sepsis, diabetic ketoacidosis
- Pancreatitis
- Alcohol intoxication
- Alcohol withdrawal
- Other toxic ingestions

Investigations

- Labs: electrolytes, glucose (low or normal), ABG (anion gap acidosis), urinalysis (ketoacids) LFTs, ◇; magnesium, serum ketones, blood alcohol (may be low as patient symptoms or mental status limits intake; may allow differentiation and detection of toxic alcohols), lactate (determine presence of a mixed anion gap acidosis) ◈
- Imaging: upright CXR (rule out perforated oesophagus or bowel) ◇

Management

The goal of acute management is restoration of fluid, electrolyte and acid-base balance, and correction of life-threatening complications. Patient fluid deficits may be significant.

- IV fluid resuscitation: will reverse the metabolic conditions resulting in ketosis. Monitor glucose carefully. Add dextrose to IVF, and treat hypoglycaemia with 10–50% dextrose as needed
- Thiamine 100 mg IV: alcoholics should receive thiamine along with dextrose-containing solutions (note that there is no evidence supporting the need to give thiamine first) ◈
- Manage any comorbid illness such as pancreatitis, alcoholic hepatitis, malnutrition, neurologic sequelae of long term alcohol abuse. Evaluate for coagulopathy and GIB

Critical documentation

Note serial VS evidence of significant alcohol ingestion, signs of alcohol withdrawal and response to therapy, glucose, ABG results, any relevant comorbidities, treatment and response.

Disposition

Admit all patients, preferably to high-level care.

159 Hyperthyroidism

Hyperthyroidism ranges in severity from a mild, asymptomatic laboratory finding to thyrotoxicosis, a severe, potentially fatal medical condition.

The first five minutes

- ABC, VS, IV, cardiac monitor

History and physical examination

Key historical features

Ask about medication history, family or personal history of thyroid disease, recent illness.

Signs and symptoms

Symptoms include anxiety, dyspnoea, tremulousness, vomiting, diaphoresis, palpitations, weight loss, diarrhoea, confusion and visual complaints.

Physical examination findings may be non-specific. Patients may have signs of chronic hyperthyroidism such as exophthalmos and hair loss. Look for: tachycardia, high output heart failure, hyperthermia, hypertension, tachydysrhythmias, tremor, enlarged or tender thyroid (single or multiple nodules), and delirium.

Possible causes and differential diagnosis

Causes of hyperthyroidism include:

- Graves' disease (50–60% of cases)
- Subacute thyroiditis
- Excess exogenous thyroid hormone intake
- Toxic thyroid adenoma
- Toxic multinodular goiter
- Amiodarone
- Post-partum hyperthyroidism

Investigations

- Labs: electrolytes, renal ◇; TSH (suppressed to near zero in severe hyperthyroidism), T3 and T4 (T3 is the

unbound form and is biologically active; measuring T3 and T4 is only necessary if TSH is normal (~5% of hyperthyroid patients)) ⟐
• ECG: ◇ atrial tachycardias
• Imaging: scintigraphy (radioactive I^{123} uptake scan; may differentiate between multinodular goiter, thyroid adenoma and Graves' Disease) ⟐
• Biopsy (nodules identified either on physical examination or via radioactive uptake scans - assess for malignancy) ⟐
• Infection is a common trigger. Always evaluate for occult infection (CXR and urinalysis), MI, or other cause

Management

The goal of acute management is treatment of acute, life-threatening complications. Patients in thyroid storm or severe thyrotoxicosis may require urgent resuscitation for life threatening dysrhythmias, severe volume depletion and/or mental status changes.
• Propranolol: ◇ 10–30 mg PO Q6h OR 1–3 mg IV at 1 mg/min initially (repeat as needed to total of 5 mg):
  » Beta blockers diminish the symptoms of hyperthyroidism and will assist in the control of tachydysrhythmias
• Methimazole: ⟐ 20–30 mg TID for short term, then reduce dosage to maintenance (5–15 mg/day) or reduce frequency to daily:
  » Inhibits T3 and T4 synthesis
• Propylthiouracil: ⟐ rapid acting, controversial due to associated liver failure. Use in severely ill patients and those unable to tolerate other therapy. 300–450 mg/day PO divided TID initially (may require up to 600–900 mg/day; maintenance dose is 100–150 mg/day divided TID):
  » Inhibits T3 and T4 synthesis and as well peripheral conversion of T4 to T3
• Inorganic iodides or iodinated contrast material:
  » Lugol's solution, SSKI, sodium $iodide^{131}$ or iopanoic acid ◇
  » Lugol's solution: 250–500 mg (5–10 gtt of 1 g/ml) PO Q6h
  » May exacerbate thyrotoxicosis from toxic adenoma and toxic goitre
• Hydrocortisone: 15–240 mg PO/IM/IV BID ◇

Critical documentation

Signs and symptoms, TSH level, evidence of cause (e.g. Graves' disease, subacute thyroiditis), infectious work up and response to treatment.

Disposition

Admit patients with severe hyperthyroidism/thyrotoxicosis, and those with tachydysrhythmias or cardiac failure, preferably to high care.

160 Hypothyroidism

Symptoms of hypothyroidism can manifest in almost all organ systems and can range from mild, subclinical disease to life-threatening myxoedema coma (typically precipitated by an acute insult, such as infection).

The first five minutes

• ABC, VS, glucose level, IVF as needed

History and physical examination

Key historical features

Ask about medication history and recent illnesses.

Signs and symptoms

Symptoms include dizziness, vomiting, lethargy, weakness, vocal changes, cold intolerance, confusion, depression, constipation, weight gain and menorrhagia or menstrual irregularity. Exam findings may be non-specific: AMS, depression, bradycardia, hypothermia, skin is dry/cool/coarse, hair loss, goitre, and periorbital oedema.

Possible causes and differential diagnosis

Differential diagnosis

The differential diagnosis for hypothyroidism is broad.
- Sepsis/infection
- Toxic ingestions
- Depression
- Encephalopathy

Possible causes

- Iodine deficiency
- Hashimoto's thyroiditis
- Surgical thyroid resection or radioactive ablation with iodine (I^{131})
- Congenital hypothyroidism
- Secondary hypothyroidism (dysfunction of hypothalamus or pituitary)
- Medications (lithium, carbamazepine, phenytoin, amiodarone, chemotherapeutics)
- Patients in myxoedema coma frequently have an inciting event such as infection

Investigations

- Labs: electrolytes, renal, glucose, ABG (hypoventilation and hypoxia in myxoedema) ◇; TSH (elevated in primary hypothyroidism), T3, T4 (T3 is metabolically more active) ⟐
- ECG: ◇ (pericardial effusion (low voltage and electrical alternans), bradycardia or conduction delays)

Management

The goal of acute management is identification and treatment of life threatening cardiac, neurologic and respiratory complications.

Mild hypothyroidism:
- Thyroid hormone (Levoxyl®, Synthroid®): 12.5–25 mcg/day PO initially; adjust dose by 25 mcg/day q2–4 week PRN

Myxoedema coma:
- Thyroid hormone (Levoxyl®, Synthroid®): 200–500 mcg IV once, then 100–300 mcg one day later PRN. Patients may be unresponsive to other emergency measures until thyroid hormone is administered

Critical documentation

Note evidence of hypothyroidism, lab results, suspected precipitating event, treatment and response.

Disposition

Admit all patients with myxoedema coma, preferably to ICU ⟐.

161 Adrenal and pituitary emergencies

Adrenal emergencies

Adrenal insufficiency is a life-threatening deficiency of steroid hormones in which the body is unable to mount a

response to physical stressors such as infection, myocardial ischaemia, pregnancy, trauma, or surgery. It may be caused by abrupt discontinuation of steroids, and presents with severe hypotension and shock.

Pituitary emergencies

The pituitary gland secretes many hormones: thyroid stimulating hormone, gonadotropins, growth hormone, prolactin, oxytocin, vasopressin, and adrenocorticotropic hormone (ACTH). ACTH deficiency is a medical emergency causing cortisol deficiency: it presents and is treated identically to adrenal insufficiency in the emergency setting. Deficiency of other pituitary hormones may present sub-acutely with signs and symptoms specific to the deficient hormone. Postpartum pituitary necrosis (Sheehan's syndrome) may present with acute onset symptoms of pituitary insufficiency and severe headache in the postpartum setting.

The first five minutes

Transfer the patient to resuscitation room if available.

- ABC, VS, high flow O_2, IV, cardiac monitor
- IVF: 2 L in first hour minimum for adults
- Hydrocortisone or dexamethasone STAT dose

History and physical examination

Key historical features

Check for history of adrenal insufficiency; recent pregnancy; headache or head injury, and steroid use (prednisone, dexamethasone, etc.). Evaluate for triggering stressor: recent surgery or trauma, chest pain suggestive of myocardial ischaemia, weakness suggestive of stroke, pregnancy, etc.

Signs and symptoms

Look for weight change, lethargy, weakness, mental status changes, syncope, and salt craving. Hyperpigmentation, cushingoid appearance. Check for gynaecomastia in men/menstrual changes in women/delayed growth and sexual maturation in children.

Differential diagnosis

The primary differential diagnosis for severe hypotension is septic shock (which also may be the trigger of adrenal or pituitary emergency). Consider GI bleed, MI, anaphylaxis, acute abdomen, haemorrhagic stroke.

Investigations

Primary adrenal insufficiency is adrenal failure: It is diagnosed by low cortisol, low aldosterone, high ACTH.

- Labs: CBC, electrolytes (classically hyponatraemia and hyperkalaemia, though electrolyte changes may be late and subtle), renal, glucose, pregnancy test, ABG, urinalysis ◇; random serum cortisol ⟐
- ECG ◇: (ischaemia)
- Imaging: CXR (pneumonia) ◇; CT head (symptoms of stroke) MRI brain (pituitary mass, when stable), contrast CT abdomen (adrenal mass, when stable) ⟐

Management

The goal of acute management for steroid insufficiency (whatever the source) is early recognition and provision of high-dose empiric steroids while awaiting hormone test results, treatment of shock with IV fluids and, if necessary, vasopressors, and diagnosis and treatment of the precipitating stressor.

- IV crystalloids: patients with adrenal insufficiency will require many litres of fluid in the first hours. Monitor for volume overload
- Broad-spectrum empiric antibiotics for possible infectious trigger or sepsis
- High-dose steroids most readily available. Dexamethasone will not interfere with adrenal testing. Hydrocortisone

provides both glucocorticoid and mineralocorticoid

- Dextrose to correct hypoglycaemia and frequent re-checks of glucose; patient may have severe hypoglycaemia
- Vasopressors ⟐: if refractory hypotension after volume resuscitation

Critical documentation

Serial VS, response to initial therapy, and dose and time of medications provided. Clearly mark time of next dose of steroids and next glucose check.

Disposition

Admit all patients, preferably to ICU ⟐, and review by endocrinologist where available. Stabilise prior to transfer: endocrine evaluation is not urgent; treatment of shock is.

162 Magnesium disorders

The first five minutes

- ABC, VS, IV, cardiac monitor
- ECG

Hypomagnesaemia

Magnesium normal range is 0.7–1.05 mmol/l (1.5–2.5 mg/l). The rate of change is more important than the absolute value.

History and physical examination

Key historical features

Ask about medication history, food intake, and alcoholism.

Signs and symptoms

Usually asymptomatic and found on blood tests, but may cause dizziness, confusion, lethargy, weakness and tremors. Look for dysrhythmias, tachycardia, hypertension, hyperactive deep tendon reflexes, positive Chvostek and Trousseau signs, and carpopedal spasm.

Possible causes and differential diagnosis

Causes include alcoholism, malnutrition, diarrhoea, medications (diuretics, proton pump inhibitors, digitalis, aminoglycosides), renal losses (acute tubular necrosis, Bartters syndrome, post obstructive diuresis) and diabetes.

Investigations

In addition to Mg levels, always check K and do an ECG. Other investigations are directed at complications and finding a cause.

- ECG ◇: (ST segment depression, peaked T waves, U waves, PR prolongation, widened QRS)

Management

The goal of acute management is to address life threatening complications and increase serum Mg. Identify and treat the underlying cause; correct hypokalaemia.

- Mild, asymptomatic cases – oral magnesium (or diet – green vegetables) magnesium oxide, magnesium gluconate: (5–7 mEq, 2.5–3.5 mmol, 60–84 mg per tablet) four tablets per day
- Severe deficiency – IV magnesium sulphate: 1 g IM Q6h up to 5 g IV over three hours depending on the patient. Maintenance: 30–60 mg/kg/day IV

Hypermagnesaemia

Hypermagnesaemia is less common and is primarily iatrogenic. The severity of disease depends on the level and the rate of change.

History and physical examination

Key historical features

Typically iatrogenic, or may have a history of renal failure.

Signs and symptoms

As for hyperkalaemia (p. 426).

Possible causes and differential diagnosis

Mainly iatrogenic (IV Mg or TPN) and renal impairment.

Investigations

In addition to a Mg level and an ECG, all patients need a K level.
- ECG ◇: (intraventricular conduction delay, PR prolongation)

Management

The goal of acute management is to address life threatening complications and decrease serum Mg. Correct hyperkalaemia.

If ECG changes:
- IV furosemide 40 mg (avoid in hypotension)
- Calcium gluconate 10 ml of 10% IV (or calcium chloride) ◈
- Dialysis for resistant cases

Critical documentation

Record serial Mg level, ECG changes, therapy and response, and suspected cause.

Disposition

Admit patients with ECG changes or needing IV therapy.

163 Sodium disorders

The first five minutes

- ABC, VS, IV
- Bedside Na level (ABG or rapid test)

Hypernatraemia

Defined as serum Na > 145 mmol/l, hypernatraemia reflects a decrease in total body water (TBW) and is usually associated with high mortality.

History and physical examination

Key historical features

Ask about the patient's medication history and recent oral intake.

Signs and symptoms

Symptoms depend on the level as well as time course over which it increased; symptoms are most notable > 158 mmol/l: confusion/agitation, vomiting, headache, dizziness, muscle twitching, fatigue, coma, seizure, thirst, anorexia, and polyuria/polydipsia (diabetes Insipidus).

Exam non-specific: AMS/coma, seizure, hyperreflexia, ataxia, muscle spasticity, signs of dehydration.

Possible causes and differential diagnosis

Can be thought of as inadequate body water, or excessive body Na.

- Inadequate water intake:
  - Inability to access water
  - Impaired thirst (inability to sense thirst or damage to hypothalamus)
  - Increased losses (diarrhoea, vomiting, burns)
- Excessive Na relative to body water content:
  - Iatrogenic
  - Cushing's syndrome
  - Diabetes insipidus (DI) (central or nephrogenic). Either lack of production of ADH or a failure of its impact on renal collecting system. Dilute urine produced and free water lost
  - Osmotic diuresis (any causes of diuresis of free water, e.g. post-obstructive diuresis, acute tubular necrosis)
  - Medications: alcohol, lithium, phenytoin, amphotericin, sulfonylureas

Investigations

- Glucose, electrolytes, renal; magnesium, serum osmolality, urine sodium, urine osmolality ◈
- Other investigations are directed at cause

Management

The goal of acute management is cautious reduction of Na and treatment of complications. Management of hypernatraemia principally involves volume replacement. Initially this can proceed with IV NS until perfusion has been restored. Reduction in Na should not exceed 10–12 mmol/l/day – rapid reduction (particularly in chronic hypernatraemia) may result in cerebral oedema.

- Hypotonic solutions can be used cautiously once volume has been restored with isotonic normal saline ◇
- Frequent monitoring of plasma Na is vital

Treat the cause.

Disposition

Admit patients with symptoms; monitor closely.

Hyponatraemia

Defined as a plasma Na < 135 mmol/l – clinical signs generally do not occur until < 125 mmol/l. Patients whose hyponatraemia has developed over long periods of time may lack symptoms. Plasma Na concentration reflects water balance in an individual and is usually tightly regulated by thirst and renal function. Overly rapid correction of hyponatraemia can result in permanent brain damage via cental pontine myelinolysis.

History and physical examination

Key historical features

Ask about the patient's medication history and oral intake in the recent past.

Signs and symptoms

Symptoms include: dizziness, vomiting, headache, muscle cramping, confusion, fatigue, coma, tremulousness or seizure, thirst and anorexia. Physical signs include: coma, seizure, dehydration, signs of cerebral oedema (late).

Possible causes and differential diagnosis

- Hypovolaemic hyponatraemia:
  - Dehydration (vomiting, diarrhoea, sweating, third spacing, hydration with hyposmolar fluids)
  - Cerebral salt wasting syndrome
  - Diuretic use
- Euvolaemic hyponatraemia:
  - Psychogenic polydipsia
  - Iatrogenic (hypotonic IV fluid, bowel preparation solutions, many medications)
  - SIADH (syndrome of inappropriate ADH secretion)
- Hypervolaemic hyponatraemia:
  - Renal failure/salt wasting nephropathy
  - Cirrhosis
  - Nephrotic syndrome
  - Congestive cardiac failure
  - Beer potomania
  - MDMA (Ecstasy use)
  - Hypothyroidism, hypocortisolism
- Pseudohyponatraemia (from significant osmotically active solutes, e.g. hyperglycaemia, hypertriglyceridaemia)

Investigations

- Labs: glucose, electrolytes, renal ◇; magnesium, LFTs, serum osmolarity, urine sodium,urine osmolarity ◈
- Imaging: upright CXR (suspected pulmonary infection) ◇, CT brain (suspected cerebral process) ◈

Management

The goal of acute management is treatment of serious complications, and careful restoration of serum sodium concentration. Management depends on the cause, severity, and time course.

In mild cases cessation of offending medications or free water restriction may suffice. It is crucially important to avoid overly rapid corrections as this may result in central pontine myelinolysis, and death.

- For severe hyponatraemia (seizures, coma, signs of brainstem herniation), consider treatment for cerebral oedema and elevated ICP ◈:
  - Hypertonic 3% saline. 3–5 ml/kg saline over 15–60 min to increase Na by ~2–4 mmol/l. The goal of hypertonic saline administration should be only to a level at which an initial rise of < 4–6 mmol/l over 1–2 hours). Then pursue more gradual correction
- Close monitoring of serum sodium initially hourly with a target correction rate of no more than 8–10 mmol/l/day, and less than 0.5 mmol/l/hour (once initial emergency correction has been accomplished if necessary)
- Hypovolaemic hyponatraemia
  - Determine Na deficit:

> Total body Na deficit = (desired Na – actual plasma Na) × TBW
> (TBW = total body water) = 0.6 × weight in kg for men; 0.5 × weight for women

  - Replace with normal saline (0.9%)
  - Restriction of free water intake may be required depending on the cause of hyponatraemia ◇
  - Correct at no more than 0.5 mmol/l per hour

Note: patients with chronic, severe hyponatraemia appear to be at higher risk for the development of central pontine myelinolysis. Monitor serum Na frequently and limit rate of correction.

Disposition

Admit symptomatic patients and those who require correction with NS.

Critical documentation

In both conditions, note symptoms, serial sodium level, likely cause, treatment given and response.

164 Calcium disorders

Calcium exists in plasma as protein bound and unbound (ionised) fractions. The ionised fraction is physiologically active; changes in serum protein levels affect the total amount of calcium in the serum but not the ionised fraction. When total serum Ca is used, it must be corrected if albumin is abnormal. Normal range: 2.2–2.6 mmol/l (8–10.5 mg/dl).

> To calculate corrected total calcium:
>
> Add 0.1 mmol/l for every 4 g/l that albumin is below 40 g/l; subtract for every 4 g/l over 40.

Hypercalcaemia

Most commonly caused by hyperparathyroidism or malignancy. Often asymptomatic and only noted on labs. Serum Ca levels > 4 mmol/l (14 mg/dl) typically result in significant symptoms.

The first five minutes

- ABC, VS, IVF; cardiac monitor
- ECG

History and physical examination

Key historical features

History of malignancy, medications, thyroid or renal problems.

Signs and symptoms

Bony or abdominal pain, vomiting, polyuria/polydipsia; renal or gall stones; constipation; depression, anxiety or other acute psychiatric disorder, hypertension, bradycardia, dehydration.

Physical examination may be non-specific and may relate to the underlying cause (e.g. malignancy).

Possible causes and differential diagnosis

The differential is broad. Causes include:

- Primary hyperparathyroidism
- Malignancy, particularly when widely metastatic (breast, lung, multiple myeloma, squamous carcinoma)
- Lithium, thiazide, theophylline, or vitamin D toxicity
- Pheochromocytoma/multiple endocrine neoplasm (MEN)
- Hyperthyroidism
- Paget’s disease of the bone
- Sarcoidosis
- Renal failure

Investigations

- Labs: electrolytes (many patients are hypokalaemic), renal ◇; TSH, T_3, T_4, parathyroid hormone, phosphate, albumin ⟐
- ECG ◇: (short QT, long PR, wide QRS, heart block; digoxin toxicity may be more pronounced)
- Imaging: CXR (look for cause, e.g. mediastinal LAN (sarcoid), lung mass (carcinoma), extremity and spine XRs (multiple myeloma or metastatic disease) ◇; nuclear thyroid and parathyroid scans ⟐

Management

The goal of acute management is volume repletion, identification of bony or cardiac complications, and symptom relief. Many patients with mild hypercalcaemia are asymptomatic and require little acute treatment.

Treat extreme symptomatic hypercalcaemia with:

- Volume repletion (IV crystalloid) ◇
- Bisphosphonate medications inhibit bone resorption (pamidronate 90 mg IV over 24 hours or etidronate 7.5 mg/kg/day IV for 3 days, zoledronic acid 4 mg IV over 15 minutes) ⟐
- Calcitonin prevents bone resorption and increases calcium excretion: 4 units/kg SC ⟐

Loop diuretics for malignancy related hypercalcaemia are no longer recommended.

Disposition

Admit patients with extreme hypercalcaemia, symptoms, or those requiring treatment beyond IV fluids.

Hypocalcaemia

Hypocalcaemia is a relatively common electrolyte disturbance defined as an ionised $[Ca^{++}] < 2.0$. Chronic hypocalcaemia may be asymptomatic. Hypocalcaemia with neurological, muscular or cardiac dysfunction is associated with significant morbidity and mortality.

The first five minutes

- ABC, VS, IVF, cardiac monitor
- ECG

History and physical examination

Key historical features

Report paraesthesia, muscle cramping, tetany, carpopedal spasm, or history of malignancy or thyroid procedures. renal, pancreas or liver disease, transfusion, medications.

Signs and symptoms

Symptoms correlate most with the degree of hypocalcaemia and the rapidity of Ca decline. Patients may have no symptoms. Look for Chvostek’s sign (muscle spasm is provoked when the facial nerve is tapped just anterior to the ear), Trousseau’s sign (muscle fasciculations and carpopedal spasm elicited by pumping a BP cuff above SBP and observing the extremity), seizure, bronchospasm, confusion, hypotension.

Possible causes and differential diagnosis

Ca is supported largely by PTH and renal modification of calcium reabsorption. Many causes of hypocalcaemia relate to dysfunction of one or both of these pathways. Causes include:

- Hypoalbuminaemia: cirrhosis, nephrotic syndrome, malnutrition
- Parathyroid absence, destruction or dysfunction: DiGeorge syndrome, surgical excision, radioablation
- Polyglandular autoimmune disease (PGA-1)
- Reduced parathyroid hormone secretion

- Hypomagnesaemia
- Vitamin D deficiency: malnutrition, malabsorption, liver disease
- Vitamin D resistance: rickets, Fanconi syndrome
- PTH resistance: pseudohypoparathyroidism
- Hyperphosphataemia
- Chronic kidney disease
- Hungry bone syndrome: corrected primary or secondary hyperparathyroidism leads to rapid sequestration of calcium
- Pancreatitis
- Medication effects: bisphosphonates, cinacalcet, phenytoin, chemotherapeutics.

Investigations

- Labs: electrolytes, renal, LFTs (acute or chronic liver disease may contribute to hypocalcaemia via hypoalbuminemia or vitamin D deficiency) ◇; magnesium, phosphate, albumin, lipase (pancreatitis may cause hypocalcaemia), PTH, vitamin D ◈
- ECG ◇: (prolonged QT, VF, heart block)

Management

The goal of acute management is identification and treatment of life-threatening complications of hypocalcaemia. The treatment depends on the cause, the degree as well as the rate at which the hypocalcaemia developed.

Patients with mild disease may be placed on oral calcium therapy and discharged with close follow up and a plan for further investigation. Hypomagnesaemia must be corrected.

- Mild:
  - » Calcium carbonate tablets: 1–2 g PO TID as necessary
  - » Calcium citrate tablets: 1 g/day divided
  - » Vitamin D (calcitriol): (chronic renal failure) initial: 0.25 mcg PO qDay to every other day; titrate by 0.5–1 mcg/day q4–8 weeks. Diet: chronic renal failure: increase dietary calcium to greater than 1 g/day and limit phosphate intake
- Severe (symptomatic, arrhythmias or hypotension):
  - » Doses of 100–300 mg of elemental Ca (10 ml of gluconate contains 90 mg elemental Ca; 10 ml of CaCl contains 272 mg elemental Ca) in 50–100 ml of 5% dextrose over 5–10 minutes. CaCl should be given only in emergencies and via central line ◈
  - » Continuous Ca infusions should be started at 0.5 mg/kg/hr and increased to 2 mg/kg/hr if necessary ◇
  - » Frequent monitoring required
  - » Once stabilised use oral therapy
  - » Note: Ca can potentiate digoxin toxicity; caution in patients taking digoxin

Critical documentation

Note calcium level and corrected calcium, symptoms, ECG findings, likely cause, treatment and response.

Disposition

Admit and monitor patients who require IV calcium and those with severe comborbid disease.

165 Potassium disorders

The normal total serum K is 3.5–5.0 mmol/l; it is primarily intracellular.

The first five minutes

- ABC, VS, IV, cardiac monitor
- Bedside K level (ABG or rapid test)

• ECG

Hyperkalaemia

Hyperkalaemia is a common problem most often due to impaired urinary excretion. Levels > 6.5 mmol/l can be life-threatening. Always recheck a high lab value.

History and physical examination

Key historical features

Ask about medication history, diet, renal disease, hypertension, fluid intake, urine output.

Signs and symptoms

Usually asymptomatic and is detected on blood tests. Moderate to severe hyperkalaemia can produce nausea, fatigue, muscle weakness, abdominal pain.

Physical examination may be normal. Bradycardia, tachypnoea due to respiratory muscle weakness and tenderness, and absent tendon reflexes may be noted. Look for flaccid paralysis. Monitor may show wide QRS.

Possible causes and differential diagnosis

Causes include spurious results (false positive due to haemolysis), decreased renal excretion (diuretics, renal failure), cellular shifts (acidosis, drugs), and cell injury (burns, rhabdomyolysis, transfusion reaction).

Investigations

• Labs: K ◇; magnesium (tends to be raised with K) ◈
• ECG ◇: (peaked T & short QT to small P, long PR and wide QRS to sinusoidal rhythm and VF)

Management

The goal of acute management is to address life-threatening complications and reduce serum K. Treat levels > 6.5 mmol/l or any elevation associated with ECG changes.
• IV NS infusion will help if acidotic
• Calcium gluconate 10 ml of 10% IV ◈ (or calcium chloride ◇) to stabilise myocardium
• 10 units of rapid acting insulin with 25–50 ml of 50% dextrose IV to shift Kintracellularly
• Nebulise 5 mg salbutamol
• Kexelate® binding resin to facilitate excretion ◈
• Dialysis for resistant cases

Note that only Kexelate and dialysis lower total body K. Other therapies shift K intracellularly.

Disposition

Admit all patients, preferably to a high care setting with a monitored bed ◈. Identify cause and evaluate future risk.

Hypokalaemia

Hypokalaemia is defined as plasma K < 3.5 mmol/l, and is life-threatening if < 2.5 mmol/l. It is of special concern in patients on digoxin.

History and physical examination

Key historical features

Ask about diet (look for eating disorders) and use of medications and laxatives.

Signs and symptoms

Tends to be asymptomatic and found on blood tests, but may cause constipation, weakness, paralysis and abdominal cramps. Signs include bradycardia, weakness, ileus and reduced tendon reflexes.

Possible causes and differential diagnosis

K may be lost from the kidneys (renal tubular acidosis, hyperaldosteronism) or gut (vomiting, diarrhoea, laxatives), be affected by drugs (steroids, diuretics). It may be a spurious result. Look for Cushing's syndrome, hypocalcaemia and hypomagnesaemia.

Investigations

- ECG ◇: (U waves (represents delayed ventricular repolarisation), flat or inverted T waves, depressed ST segment, prolonged QT (leads to SVT, AF, VT, especially in patients on digoxin))

Management

The goal of acute management is to address life-threatening complications and increase serum K. Identify and treat the underlying cause. Check for and correct hypomagnesaemia ◈.

Be very cautious in potassium replacement in the elderly, or patients with renal impairment or diabetes. A decrease in serum K of 1 mmol/l represents approximately 300 mmol total body loss.

Use oral supplementation if possible (20–40 mEq q 4 hours). For those with ECG changes, IV K at 10–20 mmol/hr (concentration should not exceed 40 mmol/l for peripheral lines or 60 mmol/l for central lines, to prevent irritation). Avoid dextrose-containing fluids to prevent intracellular shift via insulin release.

Disposition

Admit patients with ECG changes and those needing IV therapy.

Critical documentation

In both conditions: document presence of symptoms, serial plasma K, ECG changes, therapy given and any response.

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Part

4

J. Neurology

166 Approach to weakness

True weakness is defined as 'the inability to carry out a desired movement with normal force because of a reduction in strength of the muscles,' although patients may use weakness as a synonym for lethargy, dizziness, or other symptoms. The term *paresis* describes partial loss of strength, while *plegia* and *paralysis* refer to a total loss of contractility. True weakness may result from disorders of the brain, spinal cord, peripheral motor nerves, neuromuscular junction, or muscles themselves, and there are characteristic features to each. *Upper motor neurons* (UMN) connect the cerebral cortex to the spinal cord (or brainstem to cranial nerve motor nuclei), and synapse with *lower motor neurons* (LMN) that connect to muscle. Neurologic disorders are sometimes categorised by which neurons they affect. Non-neuromuscular conditions associated with generalized weakness include sepsis, MI, hypoglycaemia, electrolyte imbalance (especially K, Ca, Mg), toxicologic, and endocrine conditions, and are discussed elsewhere.

The first five minutes

- ABC, VS, O_2, IV
- Evaluate strength of respiratory effort
- Distinguish generalised (consider non-neuromuscular causes and systemic illness) from focal (see 🕮 Stroke, p. 464) weakness
- Rapid glucose; and bedside electrolyte testing

History and physical examination

Key historical features

Ask about:
- Onset and progression over time
- Distribution and progression: unilateral, bilateral, ascending GBS, tick paralysis); descending (botulism, myasthenia gravis (MG)); migratory (multiple sclerosis)
- Exacerbating or relieving factors: response to activity, Uhthoff's phenomenon (MS worsens with fever, hot bath, hot climate, exercise)
- Medical history:
  - » TB (spine/brain lesion), HIV (stroke, myelopathy, peripheral neuropathy, space occupying lesion (SOL)), DM (stroke, peripheral neuropathy), cardiovascular disease (stroke/TIA), thyroid disease
- Social history:
  - » Alcohol use (peripheral neuropathies), IV drug use (epidural abscess)
  - » Occupational/environmental exposures (lead, mercury, arsenic, pesticides, animal bites)
  - » Food: canned goods (botulism), fish/shellfish (cigua/paralytic toxins), daily cassava (tropical ataxic neuropathy)

Signs and symptoms

Anatomic localising features

UMN are contained entirely within the CNS, and lesions are associated with normal or increased tone and spasticity. Pronator drift is a subtle sign of UMN weakness. LMN transmit signals from the CNS to the muscles, and lesions are associated with decreased tone and fasciculation acutely, and muscle atrophy in the chronic phase. (See 🕮 Myotomes p. 941)
- Brain (see also 🕮 Stroke p. 464):
  - » Usually unilateral (a brain lesion large enough to cause bilateral weakness will usually also cause abnormal level of consciousness)
  - » Focal weakness + cortical signs (aphasia or neglect) → cortical stroke
  - » Crossed findings (facial weakness and contralateral body weakness) → brainstem stroke (affecting cranial nerve and descending motor tracts prior to decussation)
  - » Associated headache, nausea/vomiting, visual symptoms, seizures, cranial nerve, cerebellar function abnormalities
- Cord:
  - » Bilateral weakness of legs or arms with clear transition level and normal mental status → spinal cord lesion
  - » Unilateral motor weakness, with contralateral loss of pain/temperature → ipsilateral spinal cord lesion
  - » Associated with back pain, fever, weight loss, urinary retention, stool incontinence
- Peripheral nerves:
  - » Sensory symptoms in same distribution as weakness
  - » Localised pain
  - » History of trauma
- Neuromuscular junction:
  - » True **fatigue**, the 'diminution of strength with repetitive actions', is characteristic of neuromuscular junction disorders
- Muscle:
  - » Proximal > distal weakness is characteristic of myopathy
  - » Inflammatory myopathy may be associated with localised tenderness

Key examination components
- General:
  - » Breathing, swallowing, speaking difficulties
  - » Wasting (TB, HIV, malignancy, nutritional deficiencies)

- Motor strength testing:
  - » Control pain
  - » Check withdrawal to pain if patient is obtunded or uncooperative
  - » Check for L/R symmetry and upper/lower extremities differential
  - » Include pronator drift and heel/toe walk to test for subtle weakness
- Reflexes:
  - » Increased (± upgoing Babinksi): UMN/CNS
  - » Decreased/normal: LMN/muscle
- Sensory:
  - » Always check bilaterally as brainstem and spinal lesions may have crossed findings
  - » Check light touch and pain/temperature sensation
  - » Check for sensory level
- Musculoskeletal:
  - » Normal/increased tone, spasticity: UMN
  - » Decreased tone, fasciculation: LMN, muscle
  - » Muscle atrophy: chronic
- Vascular:
  - » Murmurs, irregular rhythm, carotid bruits: embolic stroke
- Respiratory:
  - » Weakness affecting the diaphragm may present as respiratory distress with accessory muscle use, difficulty clearing secretions: MG, GBS

Possible causes

Generalised neuromuscular weakness

Tetanus (spasticity) and diphtheria toxins, paralytic rabies.

Episodic weakness

Multiple sclerosis (symptoms vary in location and time, UMN signs, Uthoff's phenomenon); hypokalaemic periodic paralysis.

Brain

TIA/stroke, haemorrhage, SOL, MS.

Cord

Tumour, TB, epidural abscess, fracture with compression, transverse myelitis, multiple sclerosis, spinal schistosomiasis, syphilis (tabes dorsalis), spinal infarction, tropical spastic paraparesis, central disc herniation, spinal epidural or subdural haemorrhage.

- **Anterior horn**: poliomyelitis, ALS (progressive degeneration without sensory findings; mixed UMN/LMN with fasciculation, hyperreflexia, dysarthria)

Peripheral nerve

- Spinal nerve root compression from disk herniation
- Unilateral: compression neuropathies (ulnar, radial, peroneal nerves); plexopathies (sacral, brachial, lumbar); thoracic outlet syndrome
- Distal symmetric polyneuropathies: DM, HIV, GBS, vitamin deficiency, toxins (alcohol, heavy metals, cigua, tetrodo, saxitoxin, tropical ataxic neuropathy); sensory > motor

Neuromuscular junction

- MG, Lambert-Eaton, botulism, tick paralysis, organophosphate poisoning

Muscle (myopathies)

- Inflammatory (polymyositis), electrolyte-induced, alcohol/drug, muscular dystrophy, endocrine

Investigations

- Labs: CBC, electrolytes, renal, glucose, HIV, LP ◇; CK, Ca, phosphate, test for multiple sclerosis, GBS as indicated ◈
- ECG: ◇ (in stroke syndromes)
- Imaging: XR spine (C-, T-, or L-, if lesion localises to spine) ◇; CT (brain – SOL; spine – spine bony lesion), MRI (brain – multiple sclerosis; spine – abscess or cord lesion) ◈ (📖 Neuroimaging p. 734 and Spinal p. 942)
- Other tests as indicated: nerve conduction studies, electromyography, nerve biopsies ◈

167 Abnormal gait in a child

Abnormal gait in children may result from pain, diffuse or focal weakness, cerebral or cerebellar dysfunction, bone, muscle, joint or other soft tissue conditions.

History and physical examination

Key historical features

Birth, developmental milestones (regression), neurological injury/disease, weakness, stiffness, falls, time course and evolution of symptoms.

- Pain:
  - **Nocturnal**: malignancy/osteoid osteoma/growing pains
  - **After activity**: mechanical/orthopaedic
  - **At rest, with early morning stiffness**: inflammatory arthritis
  - **Extreme**: consider malignancy, infection.
  - **Growing pains**: do **not** cause persistent limping/gait changes

Signs and symptoms

- Acute or chronic? Symmetrical or asymmetrical?
- General: LAN, anaemia
  - Signs of muscle diseases (myopathy with proximal weakness)
  - Signs of neurological disease (neurocutaneous lesions, palsies)
- Neurological: higher functions, developmental assessment, cranial nerves, cerebelllar signs, muscle tone, muscle strength, reflexes, sensation
- Abdomen: hepato-splenomegaly, masses, tenderness
- Rheumatological: absence of pain does not exclude arthritis. PGALS (paediatric gait arms and legs) assessment takes under five minutes. Temporomandibular joint involvement (unable to place three own fingers in open mouth) suggests juvenile idiopathic arthritis (JIA)

Gait assessment

Normal:
- **Stance**: starts with heel strike; ends with push-off forefoot
- **Swing**: foot off the ground
- **Toddlers' gait**: broad-based with arms outstretched for balance
- **Toe walking**: up to age three
- **Flat feet**: up to age six
- **Knock knees**: up to age seven
- **In-toeing**: up to age eight

Abnormal:
- **Antalgic**: shortened stance phase on affected side (infection/arthritis/malignancy
- **Trendelenburg**: downward tilt away from affected hip (orthopaedic hip problems)
- **Steppage/equinus**: decreased foot dorsiflexion, high swing phase (neuromuscular disease/ankle arthritis)
- **Vaulting**: knee locked at stance phase end (leg length discrepancy)
- **Stooped**: bilateral flexed hips, bilateral shortened swing phase (pelvic/abdominal pathology)
- **Ataxic**: broad-based, erratic (intoxication, movement or cerebellar disorder)

Possible causes and differential diagnosis

Possible causes

- **Symmetrical**: myositis, myopathy, muscular dystrophy
- **Asymmetrical painless**: CNS lesion, limb deformity, leg length discrepancy, JIA (can be painless)
- **Asymmetrical painful**:
  - » **Systemically unwell**: tumour (osteosarcoma, leukaemia, lymphoma), infection (septic arthritis/TB/HIV), malignancy, inflammatory/rheumatic disease (e.g. systemic JIA, SLE)
  - » **Systemically well**: mechanical, trauma, hip avascular necrosis (AVN), slipped upper femoral epiphysis (SUFE), JIA

Causes of limping by age
- All: osteomyelitis, septic arthritis, NAI, lower abdominal pain (appendicitis, UTI, hernia, testicular torsion), JIA, sickle cell anaeamia, TB arthritis, fractures
- 0–3: congenital hip dysplasia, neuroblastoma
- 4–10: transient synovitis, Legg-Calve-Perthes disease (LCPD), leukaemia
- 10–16: SUFE, primary bone tumours, Osgood-Schlatter, Sindig-Larsen-Johanssen

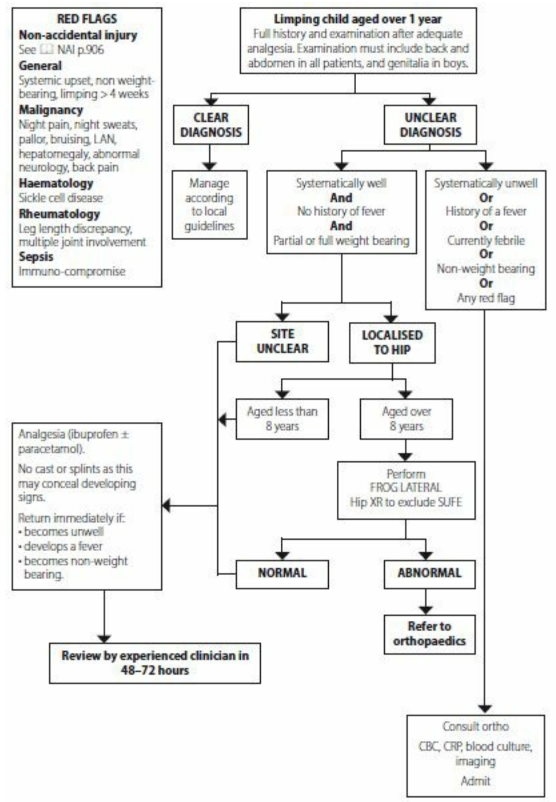
Specific conditions
- **Transient synovitis (reactive arthritis)**: common cause of hip pain in well child following viral infection. Must differentiate from septic arthritis. NSAID responsive. Typically resolves by six weeks
- **Septic arthritis**: extremely painful. Predictive: acute onset (< 5 days), fever, ESR > 35
- **JIA**: arthritis in child < 16 years lasting > 6 weeks without other identifiable cause. Oligoarticular, polyarticular, or systemic (fever, skin rash, organ involvement)
- **SUFE**: posterior/inferior displacement of femoral epiphysis. Risk factors: male, adolescent, obesity. **LCPD**: osteonecrosis femoral head epiphysis. Risk factors: male, physically active. **Both**: hip pain,limp; need urgent orthopaedic referral
- **Osgood-Schlatter** and **Sindig-Larsen-Johanssen** syndromes (tender apophysitis of the tibial tuberosity and inferior patellar pole, respectively) respond to rest/NSAIDS

Investigations

Guided by clinical findings (see algorithm); exclude serious conditions.
- Labs: CBC, ESR, HIV, TB work-up ◇; LDH, uric acid, CK, rheumatoid factor (only 10% JIA are positive)◈
- Imaging: XR spine/limb (bone lesions; early arthritis – usually normal; add frog-leg view for hip); US (joint effusions) ◇

Management

See algorithm.



168 Approach to memory loss

Some decrease in memory is an inevitable part of ageing. Inappropriate forgetfulness may be due to progressive conditions (Alzheimer's or vascular dementia), reversible causes (depression, some organic encephalopathies), toxin exposure, metabolic derangement, traumatic injury, psychiatric conditions, and inflammatory/infectious disorders.

The first five minutes

- VS usually stable; assess and address ABC as needed
- Measure blood glucose

Clinical features

Key historical features

- Acute vs chronic onset (acute – more likely to be organic disease), progressive or fluctuating etc.
- Associated symptoms – fever, weight loss, or focal neurologic deficits, seizures
- Medical history – chronic diseases (particularly vascular disease, HIV), medications changes or interactions, recent injuries and toxic exposures
- Mental illness
- Family history

Signs and symptoms

Complete examination to look for indicators of an organic cause (deranged VS, fever, trauma, complete neurological examination, evidence of atherosclerotic disease or toxidromes). Full evaluation for delirium (📖 p. 442). Mental state and psychiatric evaluation.

Differential diagnosis

- Age-related dementia
- Alzheimer's dementia or other degenerative CNS diseases
- Stroke or multi-infarct dementia
- HIV dementia
- Tertiary syphilis
- Hypothyroidism
- Vitamin deficiency (B12, folate, thiamine/Korsakoff's psychosis)
- Seizure
- Transient global amnesia (cannot recall recent events or store new memories. Usually lasts 6–24 hours with full recovery)
- Toxins – lead, alcohol
- Infection – encephalitis, meningitis, brain abscess (acute, but seldom present with isolated memory disturbance)
- Psychiatric (e.g. depression)
- Space-occupying lesions in brain (± bleed) can lead to a variety of waxing and waning neurologic deficits
- Trauma, remote or acute (📖 Head injury p. 755)

Investigations

Investigations are used to diagnose or rule out treatable causes.

- Labs: urinalysis, CBC, electrolytes, glucose, HIV, LP ◇; TSH, B12, folate
- ECG ◇: (pro-embolic arrhythmias e.g. AF)
- Imaging: CXR (TB, malignancy) ◇; CT ◈

Management

The goal of acute management is to rule out organic and reversible causes of memory loss. Management varies by underlying cause.

Critical documentation

Record a full neurologic examination, VS.

Disposition

Admit as per underlying condition.

169 Delirium

Delirium is AMS caused by a general medical condition; it is also known as 'acute confusional state'. To make the diagnosis, use the delirium assessment tool (delirium is present if features 1 AND 2 are present with EITHER 3 OR 4).

1. Acute onset (hours to days) and/or fluctuating course
2. Inattention
3. Disorganised thinking
4. ALoC

Delirium is a symptom, not a cause, and should prompt a search for the underlying condition. It may be caused by almost any illness, including:

- Infections (CNS infections, sepsis, local infections in elderly)
- Severe metabolic derangement (electrolyte, acid base and glucose)
- Drug, medication and poison ingestion

Psychiatric illness and dementia are excluded from this definition.

Delirium is a serious presenting complaint and, especially if poorly assessed and managed, is associated with significant morbidity and mortality.

The first five minutes

- ABC, VS, O_2, IV, glucose level, cardiac monitor
- Look for evidence of trauma, sepsis or other life threatening conditions

History and physical examination

Typically acute onset of fluctuating inattention and AMS (p. 576). Evaluation aims to identify the cause.

Key historical features

Delirium:

- Onset, fluctuation, content (confusion, poor attention, acute memory deficits, aggression, somnolence, personality change, behaviour change, hallucinations, aggression). Note baseline mental status

Features that could suggest the cause:

- Fever, trauma, headache, alcohol/drug use or withdrawal, industrial exposure

Features that may indicate an alternative diagnosis:

- Dementia and psychiatric illnesses usually present less acutely, are more progressive and are not typically associated with inattention and a decreased level of consciousness

Patient:

- Pre-existing medical conditions, medication, allergies, etc.
- Ask about immune compromise (particularly HIV) and malignancy

Signs and symptoms

Perform a thorough physical exam, looking for any indication of the underlying cause.

- General:
  - » Well vs. ill-appearing
  - » Fever, signs of sepsis
  - » Features of chronic illness (TB, HIV, malignancy, nutritional deficiencies)
  - » Jaundice, anaemia (hepatic encephalopathy)
  - » Features suggesting specific toxidromes
- Systemic:
  - » Any evidence of sepsis, local infections (meningitis, pneumonia, UTI)
  - » Any focal deficit suggesting stroke, IC bleeds or malignancy
  - » Any signs of trauma
- Cognitive testing (for subtle confusion):
  - » Mini-mental status examination: sensitive and specific, but time consuming
  - » MinCog test: three item recall (immediate and delayed after 3 min) and clock-drawing (p. 938). Impaired immediate recall suggests impaired attention (delirium). Intact immediate recall but impaired delayed suggests impaired memory (dementia)

Possible causes and differential diagnosis

Possible causes

Infectious:

- Meningitis, encephalitis, sepsis, brain abscess, tertiary syphilis

Endocrine/metabolic:

• Hypoglycaemia, hypoxia, hyponatraemia, hypercalcaemia, thyrotoxicosis, hypothyroidism, hypercarbia, hepatic failure, renal failure, Wernicke's encephalopathy

Cardiovascular:
• Hypotension/shock, hypertensive encephalopathy, myocardial infarction

Neurological/intracranial causes:
• Concussion, increased ICP, space-occupying lesion, stroke, vasculitis, traumatic/hypertensive/subarachnoid haemorrhage, seizures (post-ictal, non-convulsive status epilepticus, eclampsia)
Toxic:
• Intoxication and withdrawal can present as delirium
• Alcohol, benzodiazepine withdrawal, toxic alcohols, anti-epileptic drugs, anticholinergic, lithium, serotonin syndrome, neuroleptic malignant syndrome, opiates, organophosphates etc.

Differential diagnosis

Psychosis (delirium mimic)

Psychosis: normal VS neurologic exam, orientation, auditory hallucinations.

Delirium: abnormal VS and neurologic exam, fluctuating course, variable alertness, abnormal orientation and attention, visual hallucinations.

Dementia: more likely elderly, sub-acute to chronic onset, chronic progressive course, memory prominently involved, normal level of consciousness.

Investigations

All patients need blood glucose, urinalysis, ECG, CXR. Pregnancy test where appropriate.

Other tests should be guided by the suspected cause.
• Labs: CBC, electrolytes, renal, HIV, LFTs, ◇; INR, osmolality, drug levels ⟐
• ECG ◇: (MI and toxic/metabolic clues)
• Imaging: CXR ◇; CT brain ⟐

Management

The goal of acute management is to identify and address underlying causes, while preventing harm.
• Supportive care: side rails, family or staff at bedside 24/7
• Treat agitation as needed:
  » Antipsychotics (e.g. haloperidol) and/or
  » Benzodiazepines (toxicity, alcohol withdrawal, seizures)
• Find and reverse the cause

Critical documentation

Presentation, examination findings, investigation results and management and disposition plan.

Disposition

Admit patients whose acute confusion does not resolve during emergency evaluation (most patients).

170 Peripheral neuropathies

Disease processes affecting the peripheral nerves lead to motor, sensory or autonomic dysfunction. Manifestations are widely variable, and reflect the specific nerve involved and the type of involvement. By far the most common of the many causes are toxic/metabolic (particularly diabetes and alcohol abuse), inflammatory (GBS) and trauma.

The first five minutes

- ABC, VS, IV if low BP
- Check for adequate respiratory strength
- Search for signs of acute or CNS lesions (language difficulty, cognitive dysfunction, vision changes, hyper-reflexia, extensor plantar reflex) and autonomic instability

History and physical examination

Key historical features

- Onset, location, progression and course
- Precipitating event: trauma, illness, toxin exposure
- Predisposing illness (diabetes mellitus, alcohol abuse, malnutrition) and occupational exposure (heavy metals)

Signs and symptoms

- Motor: weakness with decreased reflexes
- Sensory: numbness, tingling, dysaesthesia, pain; decreased sensation to light touch, vibration, proprioception and/or temperature
- Mononeuropathy: single peripheral nerve distribution, usually from compression but may be caused by inflammation or metabolic effects:
  - » Radial: minimal numbness; wrist extension weakness ('Saturday night palsy' from laying on outstretched arm)
  - » Ulnar: medial 1½ digits numbness, hand interossei weakness (compression at elbow)
  - » Median: lateral 3½ digit numbness, weak grip, thenar muscle atrophy ('carpal tunnel syndrome')
  - » Peroneal: lateral leg numbness below knee, weakness of dorsiflexion/foot eversion (compression at lateral knee from crossing legs)
- Radiculopathy: shooting pains, numbness, and weakness in radicular pattern (examples: L4 dermatome, 'sciatica')
- Plexopathy: pain, numbness, and/or weakness in single limb that does not follow mononeuropathy, radicular, or polyneuropathy pattern
- Polyneuropathy: bilateral, symmetric sensory > motor loss in the feet ± hands ('stocking and glove'). Usually caused by toxic/metabolic conditions and inflammatory or demyelinating neuropathies

Possible causes and differential diagnosis

- Mononeuropathy: trauma, compression, early polyneuropathy
- Radiculopathy: nerve root compression (herniated disc, neoplasia, abscess, osteomyelitis, spinal TB, spinal stenosis)
- Plexopathy: (rare) DM, trauma, viral, congenital; *thoracic outlet syndrome* (position dependent symptoms (numbness with arms raised), often from compression from cervical rib, ±vascular compromise); *complex regional pain syndrome* (single limb with pain, numbness, oedema, altered skin temperature, reduced range of motion; causes: old trauma, vascular disease, DVT, rheumatoid arthritis); *plexitis* (brachial most common ('Parsonage-Turner syndrome') – numbness/weakness with deep, neuropathic shoulder pain, possible viral cause)
- Polyneuropathy: systemic (DM, vitamin B12 deficiency, hypothyroidism); infectious (HIV, CMV, leprosy); toxic exposures (alcoholism, ARVs (NRTIs like ddC, d4T, ddI > protease inhibitors), amiodarone, vincristine, dapsone, INH, ethambutol, metronidazole, nitrofurantoin, arsenic, diphtheria, organophosphates, mercury)

Danger signs

- Any respiratory abnormalities, abnormal speech or swallowing, systemic illness or change in mental state
- Ascending, symmetric pure paralysis with areflexia: GBS or tick paralysis; GBS often associated with autonomic dysfunction (haemodynamic instability, urinary retention)
- Descending, symmetric pure paralysis with areflexia: botulism (frequently associated with gastroenteritis-like

symptoms)
- Fatigable diplopia, dysarthria, dysphagia, shortness of breath, and/or proximal limb weakness: myasthenia gravis (MG) crisis

Investigations

Many patients require no investigations; tests should be directed to the underlying cause.

Polyneuropathy

- Labs: HIV, glucose ◇; B12 TSH, serum protein electrophoresis (monoclonal gammopathy) ◈
- Spirometry ◈: (MG, GBS, botulism) (danger if < 15 ml/kg)

Suspected GBS

- Labs: LP (rule out acute HIV, CMV, syphilis, sarcoidosis, TB meningitis, CNS leukaemia; typical GBS CSF: elevated protein (> 45 mg/dL) with normal cell counts (< 10 cells/mm^3)) ◇
- Spirometry ◈: (danger if < 15 ml/kg)
- Electromyogram ◈

Suspected MG

- Tensilon test (edrophonium 2 mg IV then one minute later 8 mg IV) leads to clinical improvement. Stop immediately if ↑secretions, bradycardia, vomiting
- Spirometry ◈: (danger if < 15 ml/kg)
- Electromyogram ◈

Management

The goal of acute management is to identify and treat dangerous complications (particularly respiratory insufficiency), and to identify the cause.

Mononeuropathy

- Minimise further compression
- Peroneal: splint ankle at 90° to avoid permanent foot injury

Polyneuropathy

Address cause: DM, pyridoxine (Vit B6) deficiency in isoniazide-related neuropathy.
- Pain: consider gabapentin (starting at 300 mg TID) ◈ or amitriptyline (starting at 25 mg nightly at bedtime) ◇. Avoid narcotics
- GBS, MG, botulism:
  - Monitor respiratory effort every hour
  - Assist ventilation as indicated
  - If aspiration risk, NGT feeding
- MG: pyridostigmine 60–120 mg Q6h for ongoing care (not acute management) ◈
- GBS/MG: IVIG 1 000 mg/kg IV QD × 3 days or plasma exchange 200 ml/kg over 3–5 days ◈

Disposition

Admit all patients with breathing problems, speech or swallowing abnormalities and those who cannot walk.

171 Facial palsy or droop

Unilateral facial weakness may be caused by pathology at any point from the cerebral cortex to the upper or lower tracts of CN 7. Central causes such as stroke usually present as unilateral facial weakness with relative sparing of the

forehead due to partial crossover of motor innervation fibres distal to the lesion. Lower motor neuron paralysis presents with dense unilateral weakness including the forehead and indicates pathology of the nerve itself.

Both may be associated with serious conditions (stroke, tumours) and sequelae (inability to close the eye and loss of facial cosmesis).

The first five minutes

VS, ability to protect airway, rapid neuro exam.

Possible causes and differential diagnosis

Central causes:

- Stroke, CNS bleeds, CNS tumours, trauma

Peripheral (LMN) causes:

- Bell's Palsy (acute idiopathic LMN paralysis), local infection with nerve damage (otitis, meningitis, sinusitis, parotitis, facial abscess), trauma, malignancy (brainstem, parotid, acoustic neuroma etc.), viral infection (HSV – Ramsey Hunt syndrome, EBV), other unusual causes (Lyme disease, leprosy, toxic and metabolic neuropathies)

History and physical examination

It is critical to consider treatable causes such as stroke, malignancy, trauma and local infections.

Key historical features

Sudden onset (over hours typically) unilateral facial; weakness, loss of forehead muscle tone and incomplete eyelid closure. Weakness maximal within a week. Associated features may include tearing of the eyes, abnormal taste, facial dysaesthesia, hyperacusis and a viral prodrome.

Signs and symptoms

Physical examination should search for evidence of a cause for the palsy (thereby excluding Bell's palsy) and grade the severity and complications.

- Careful neurological examination to look for other deficits that may imply stroke or CNS malignancy. Note that a small percentage of the population do not have crossover of motor innervation, meaning that a central cause may affect the forehead. All patients at high risk for vascular pathology and those with any additional neuro findings should be evaluated for stroke (📖 p. 464). Examine parotids for masses. Examine ears – infection, vesicles, masses
- Look for complications: the eye – can it close, is there a corneal ulcer? The mouth – can the patient speak, swallow and close the mouth?

See 📖 p. 933 for details of how to examine CN7.

Investigations

Bell's palsy is a clinical diagnosis. Consider HIV, syphilis and leprosy based on the clinical findings. Measure glucose.

Management

The goal of acute management is to identify central causes and minimise complications.

- Manage any cause if found: e.g. stroke (📖 p. 464), intracranial haemorrhage (📖 SAH p. 468, 📖 Head injuries p.

755)
- For Bell's (idiopathic LMN) palsy:
  - Steroids, if started early, increase probability of full functional recovery (prednisolone 30–60 mg PO daily for 7 days)
  - Currently no evidence supports use of antiviral drugs (e.g. acyclovir)
  - Eye care is important: saline or lubricating eye drops, night-time eye patch, regular eye exams. If available, facial rehabilitation exercises may speed recovery

Disposition

Discharge and follow-up patients with Bell's palsy. Complete recovery can be expected in ~85% of cases (usually by three months). Some patients may have varying degrees of permanent disability. Disposition of other patients depends upon aetiology.

172 Non-traumatic headache

Primary headaches have no clear underlying cause and comprise up to 90% of headaches in clinical practice. Secondary headaches result from an underlying identifiable pathologic process (i.e. tumours, aneurysms, meningitis, and other organic illnesses) for which the headache is the presenting symptom.
See 🕮 p. 68 for a discussion of undifferentiated and secondary headache.

Primary headaches include:
- Tension-type headache
- Migraine
- Cluster headaches
- Other (a group of rare primary headache syndromes)

The first five minutes

See 🕮 RAP Headache p. 68.

History and physical examination

See Table 172.1 for features of common primary headaches.

Possible causes and differential diagnoses

The vast majority do not represent dangerous pathology. Identify and treat dangerous headache causes (CNS infections, CNS bleeds or stroke, CNS tumours).
See Table 172.1 for details.

Danger features (red flags):
- Extremes of age (children RARELY complain of headache)
- Progressive, not responding to treatment
- AMS or LoC
- Neck stiffness or other features suggesting meningitis
- Focal neurologic signs
- First or worst headache of life
- Increasing frequency and severity of headache
- Severe headache that is maximal at onset
- Papilloedema or other features of raised ICP
- Head trauma
- Fever, cancer, or immunosuppression
- Rash suspicious for meningococcemia

• Triggered by exertion, sexual activity, or valsalva

Atypical or complex migraines may mimic features of dangerous secondary headaches:
• Ophthalmoplegic migraine: commonly with ocular nerve palsies
• Hemiplegic migraine: motor and sensory disturbance that mimics stroke
• Basilar-type migraine: brainstem effects such as vision changes, vertigo, dysarthria, paresis, and AMS
• Status migranosus: severe headache lasting more than 72 hours

Investigations

Investigations should be reserved for exclusion or investigation of secondary headaches.

Management

The goal of acute management is to rule out life-threatening secondary headaches and to relieve discomfort. Refer to Table 172.1 for details.

Migraine

• Simple measures: dark, quiet room; consider IV saline load; consider O_2 for cluster headache
• Abortive therapy: NSAID analgesia, prochlorperazine 10 mg IV ◇; if ineffective – metoclopramide 10 mg IV ◇ OR DHE 1mg IV ◈ over 3 minutes OR triptan therapy (sumatriptan 6 mg SC) ◈
• Rescue analgesia: opiates
• Drugs for headache prophylaxis have NO role in emergency management

Critical documentation

Clearly document the presence or absence of red flags.

Disposition

Admit for refractory pain or if dangerous cause identified.

Table 172.1 Differential diagnosis and treatment of most common primary headache disorders

Feature	Tension-type	Migraine (with or without aura)	Cluster
Location	Bilateral	Unilateral or bilateral	Unilateral (around or above the eye and along the side of the head/face)
Quality	Pressing/tightening (non-pulsating)	Pulsating (throbbing or banging in young)	Variable (sharp, burning, throbbing, or tight)
Intensity	Mild or moderate	Moderate or severe	Severe or very severe
Effect	Permits routine activities	Interferes with routine activities	Restlessness or agitation
Other	May be associated with neck or jaw pain	Sensitivity to light, sound nausea, vomiting Aura Classic symptoms can occur with or without headache and: • Are fully reversible • Develop over at least 5 minutes • Last 5–60 minutes Typical aura symptoms: • Visual flickering, spots, or lines and/or partial loss of vision • Sensory symptoms such as numbness and/or pins and needles and/or speech disturbance • Complex migraine: no consensus on definition	On the same side as the headache: Red and/or watery eye Nasal congestion and/or runny nose Swollen eyelid Forehead and facial sweating Constricted pupil and/or drooping eyelid
Duration	30 minutes to continuous	4–72 hours in adults 1–72 hours in adolescents	15 min–3 hours
Frequency	Episodic: < 15 days per month Chronic: > 15 days per month for more than 3 months	Episodic: < 15 days per month Chronic: > 15 days per month for > 3 months	Episodic: 1 every other day to 8 per day, with remission > 1 month Chronic: 1 every other day to 8 per day, with no remission > 1 month in a 12-month period
Feature	**Tension-type**	**Migraine (with or without aura)**	**Cluster**
Treatment	1 Quiet room, hydration, paracetamol and/or NSAIDs 2 Parenteral anti-emetics: effective for headache even when no nausea		
	DHE or triptans if refractory	Triptans or DHE Steroids for symptoms	High flow O_2 may be helpful

Adapted from: National Institute for Health and Clinical Excellence (2012) Adapted from *CG 150 Headaches: diagnosis and management of headaches in young people and adults*. London: NICE. Available from www.nice.org.uk/guidance/CG150

173 Syncope

Syncope is a transient, self-limited LoC caused by bilateral cerebral or reticular activating system hypoperfusion, usually due to loss of postural tone. In true syncope, autonomic regulation and reclining posture results in rapid and complete spontaneous recovery with no new neurologic deficits. Presyncope is near LoC and is managed as syncope. See 📖 RAP Syncope p. 78.

The first five minutes

ABC, VS, O_2, monitor, blood glucose.

History and physical examination

Key historical features

• Pre-event (prodrome (e.g. vision changes, warmth)); event (seizure activity and duration, injury, tongue biting, incontinence, duration); post-event (confusion, lethargy); associated symptoms (headache, chest pain, palpitations, dyspnoea); witnesses
• Age, structural/coronary heart disease, dysrhythmias, prior syncopal episodes

- Family history of sudden/early death
- Medications (new, changes, cardiovascular meds, QTc prolonging), illicit drugs, traditional remedies

Signs and symptoms

- VS: bilateral upper extremity BP/pulses (aortic dissection, subclavian steal) Orthostatic VS are not sensitive or specific as a volume status evaluation; many elderly have abnormal orthostatic changes at baseline
- Cardiopulmonary (murmurs, CHF findings), neurologic (new deficits), neck (signs of CHF), rectal (if GI bleed suspected), oral (lateral tongue biting suggests seizure), abdominal (AAA, peritonitis)

Possible causes and differential diagnosis

Neurovascular

- Posterior cerebral circulation deficit or transient bilateral hemisphere insult
- Vertebrobasilar TIA/insufficiency (decreased vertebral/basilar arterial flow), subclavian steal (use of arm causes retrograde flow from posterior circulation subclavian stenosis) (SAH, ask about headache after syncope)

Neurally-mediated (reflex)

- 25–65% of syncope, excellent prognosis
- Self-limited bradycardia and/or vasodilation causes transient hypotension, often with prodrome of nausea, tunnel vision, warmth, diaphoresis:
  - » Vasovagal: emotional distress, painful stimulus
  - » Situational: micturition, defecation, cough/sneeze, post-prandial, post-exercise
  - » Carotid sinus hypersensitivity: turning neck or external pressure to neck causes reflex syncope (history of tight collar, shaving, turning of head)

Cardiac

- Insufficient cardiac output (arrythmia, failure) causes LoC
- Suggested by history, physical findings, abnormal ECG
- Life threatening

Differential diagnosis

Seizure, arrythmia, CCF, metabolic (hypoxia, hypoglycaemia, hypocapnia), intoxication, cataplexy (daytime sleep disorder), drop attacks (sudden loss of tone without LoC), falls, psychogenic pseudosyncope (loss of tone, no EEG abnormalities), vertigo, head injury.

Investigations

Testing depends on clinical picture, and may include:
- Labs: pregnancy, haematocrit, glucose ◇
- ECG ◇: ischaemia, dysrhythmias, structural abnormalities

Management

The goal of acute management is to address life-threatening causes.
- Differentiate from mimics: brief convulsive movements without postictal period may accompany syncope
- Rehydration trial appropriate for suspected dehydration
- High risk indicators:
  - » Abnormal ECG
  - » History of dysrhythmia, structural heart disease, CHF
  - » SBP < 90 mmHg
  - » Dyspnoea

» Hgb < 10
» Old age, comorbidities
» Family history of sudden death
» Exertional syncope (SAH, obstructive cardiomyopathy)

Critical documentation

Event details,, VS, ECG, Hgb, pregnancy status, cardiac and neuro exam.

Disposition

Admit if high-risk cause suspected; discharge if young, low risk, no heart disease, story consistent with reflex syncope.

174 Transverse myelitis

Transverse myelitis is an inflammatory disorder that involves a complete transverse segment of the cord. Clinical manifestations are as for spinal cord compression or transection (p. 460), with disturbances of motor, sensory, autonomic and sphincter function. It usually affects young adults and has variable onset, severity and prognosis.

The most common causes are auto-immune disorders, multiple sclerosis and viral infections.

The first five minutes

Assess and address ABC as needed. Measure blood glucose.

History and physical examination

Key historical features

Ask about paraesthesia, sensory loss, weakness and sphincter dysfunction; risk factors or pre-existing disease (vascular disease, SLE, multiple sclerosis, HIV, syphilis etc.), and trauma.

- Onset is variable. Time course to peak deficit may suggest the cause:
  » < 4 hours: vascular myelopathy
  » 4 hours–21 days: consistent with transverse myelitis (differential diagnosis below); up to ⅔ reach maximal deficit by 24 hours
  » > 21 days: hereditary myelopathy, spinal cord tumour, myelopathy due to a dural arteriovenous fistulas, or a chronic progressive form of multiple sclerosis
- Urinary disturbances (retention or incontinence), constipation, sexual dysfunction
- Midline back pain or pain in a dermatomal distribution
- Symptoms of infection (rash, immunocompromised state, recurrent genital infection, endemic area for parasitic infection, TB) or systemic inflammatory disease (rash, mucosal ulcers, xerostomia, Raynaud's disease)

Physical examination

- Neurologic examination: bilateral (not necessarily symmetric) motor and autonomic spinal cord dysfunction; weakness progressing to paresis, hypertonia, hyperreflexia, clonus, and a Babinski's response; clearly defined sensory level; Lhermitte's sign (paraesthesia that radiates down the spine or limbs with neck flexion); paroxysmal tonic spasms (involuntary dystonic contractions of limb or trunk muscles). Cranial nerve deficits are rare.
- Signs of:
  » Infection (fever, meningismus, zoster, adenopathy)
  » Systemic inflammatory disease (adenopathy, serositis, inflammatory arthritis, erythema nodosum, conjunctivitis, anaemia /leukopaenia/thrombocytopaenia)

Possible causes and differential diagnoses

• Inflammatory multiple sclerosis, SLE, post vaccination
• Infections: viral (EBV, CMV, HIV etc.), syphilis, mycobacterial infections
• Paraneoplastic (malignant spinal compression is a separate entity)
• Vascular: spinal artery occlusion with cord infarction

The differential includes any causes of neurological dysfunction and sphincter disturbance. Important causes include: trauma, spinal cord compression (📖 p. 460), stroke, vertebral collapse or disk herniation with extrusion into the spinal canal, malignancy.

Investigations

Investigation of choice is MRI. Further tests are guided by clinical picture.

• Labs: glucose, LP (CSF pleocytosis, negative gram stain, normal glucose, 50% with elevated protein) ◇; serology (viral, bacteria, parasitic infection; evaluation for autoimmune aetiologies; neuromyelitis optica immunoglobulin G (NMO-IgG) antibodies) ◈
• Imaging: CXR, spinal XR ◇; CT myelography, MRI ◈

Management

The goal of acute management is prompt exclusion of treatable causes, particularly spinal compression, and symptomatic treatment.

General management includes bladder and bowel care and careful nursing to prevent bedsores. Further management depends on the cause.

Auto-immune:

• Corticosteroids are controversial. Some authors recommend methylprednisolone 0,5 g–1 g IV × 3 days ◈ or high-dose oral prednisone (1 250 mg) 1 gram daily for 3–7 days (limited evidence) ◇
• Plasma exchange (if refractory to corticosteroids) ◈

Disposition

Admit all patients to neurology (if available); physical and occupational therapy, where available.

175 Cauda equina syndrome

Cauda equina syndrome (CES) results from compression of lumbosacral nerve roots below the level of the conus medullaris, resulting in a characteristic pattern of neuromuscular and urinary symptoms. CES can lead to dramatic clinical sequelae for the patient if the diagnosis and treatment are delayed.

Causes include:

• Herniated nucleus pulposus
• Spinal infections; Pott's disease, HIV/AIDS, HSV, meningitis, syphilis, CMV, schistosomiasis, epidural abscess, *Toxoplasma* encephalitis
• Spinal trauma
• Neoplasms and metastases (breast classically), astrocytoma, neurofibroma, lymphoma and meningioma

Patients with unilateral deficits have a better prognosis than bilateral deficits; patients with bowel dysfunction typically have poor outcomes.

The first five minutes

• Typically not unstable; assess and address ABC as needed
• Analgesia

History and physical examination

Key historical features

• Variable onset with regard to speed and acuity
• Weakness, numbness or pain in the lower extremities; **bilateral sciatica**
• Altered sensation; **saddle anaesthesia** (hallmark – loss of sensation or a strange sensation in the perineum, intergluteal and inner thigh area)
• **Painless urinary retention** with overflow incontinence (may present as ↑night time urination)
• Bowel incontinence

Ask about features that may suggest the cause: back pain, weight loss, constitutional symptoms, recent trauma or surgery.

Signs and symptoms

Any combination of lower limb neurological deficit, sphincter abnormalities, back pain and abnormal perineal sensation = CES until proven otherwise.

Urinary retention > 500 ml alone or bilateral sciatica plus subjective urinary retention or rectal incontinence are strong predictors.

Local examination of the spine may reveal tumours or bony deformity.

Table 175.1 Symptoms associated with nerve root involvement

Nerve Root	Pain	Sensory deficit	Motor deficit	Reflex deficit
L2	Anterior medial thigh	Upper thigh	Slight quadriceps weakness; hip flexion; thigh adduction	Slightly diminished suprapatellar
L3	Anterior lateral thigh	Lower thigh	Quadriceps weakness; knee extension; thigh adduction	Patellar or suprapatellar
L4	Posterolateral thigh, anterior tibia	Medial leg	Knee and foot extension	Patellar
L5	Dorsum of foot	Dorsum of foot	Dorsiflexion of foot and toes	Hamstrings
S1-2	Lateral foot	Lateral foot	Plantar flexion of foot and toes	Achilles
S3-5	Perineum	Saddle	Sphincters	Bulbocavernosus; anal

Differential diagnosis

Any disease process affecting the spinal cord or peripheral nerves could present with features similar to CES.
• Progressive multifocal leucoencephalopathy
• Amyotrophic lateral sclerosis
• Diabetic neuropathy
• GBS
• HIV/AIDS neuropathies and ART-associated neuropathy
• Multiple sclerosis
• Human T cell lymphotropic virus type 1 (HTLV-1)

Investigations

The investigation of choice is spine MRI. Further investigations should be directed by the clinical condition.

Management

The goal of acute management is early recognition and referral to a spinal surgery service for intervention. Once sphincters become involved decompression surgery is critical, and morbidity increases over hours.

Provide analgesia as needed.

Critical documentation

Document history, clinical findings and investigation results.

Disposition

Admit all preferably to a spinal surgery service. ◈

176 Spinal compressive lesions

Any lesion compressing the spinal cord may result in motor, sensory, autonomic and sphincter abnormalities relating to the level and type of compression. The abnormalities may be unilateral, bilateral or follow one of many unusual spinal cord syndrome patterns. These include cauda equina (📖 p. 458) and conus medullaris syndromes.

The most common cause is malignancy (primary or metastatic); other important causes include infection (epidural abscess) and mechanical (trauma, haematoma, disc prolapse).

Rapid recognition and referral to a spinal surgeon is critical to prevent permanent disability. The diagnostic modality of choice is MRI.

The first five minutes

ABC, VS, O_2, IVF; evaluate strength of respiratory effort.

History and physical examination

Signs and symptoms

Assess for current or recent fever. Spinal examination for tenderness or deformity. Complete neurological exam with particular attention to sensory and sphincter assessment. Systemic exam may reveal features of malignancy.

See Table 176.1.

Investigations

The investigation of choice is MRI. Other tests guided by clinical picture.

Differential diagnosis

See Table 176.1.

Management

The goal of acute management is prompt recognition of compression and referral to a spinal service.

- Spinal cord compression is a true emergency, as degree of disability is often fixed at 24–72 hours. Severe or progressive neurologic deficits require emergency surgical decompression
- Provide adequate analgesia early
- Steroids are the mainstay of non-operative treatment: despite limited evidence, steroids are recommended (dexamethasone 10 mg stat initial dose)

Critical documentation

Risk factors; VS; detailed neurological examination; progression of deficits.

Disposition

Admit all to a spinal service (where available).

Table 176.1 Distinguishing features

Aetiology	Pathophysiology	Clinical features	Diagnostic aids	Management
Neoplasm	90% metastatic (lung, prostate, breast, renal cell, lymphoma)	Known cancer, pain worse when supine; 60% thoracic, 30% lumbosacral	XR for bony changes MRI is definitive	Corticosteroids, emergency radiation therapy, surgical decompression
Central disc herniation	Central herniation of nucleus pulposus impinges cord	Acute onset, associated with minor trauma; pain improved when supine	XR may show indirect signs; CT good; MRI is definitive	Corticosteroids, surgical decompression
Spinal epidural abscess (SEA)	Most often bacterial (*S. aureus*, gram negative rods, treptococci), but also fungal (aspergillosis)	Risk factors: Alcohol abuse, HIV/AIDs, trauma, immunocompromise, diabetes mellitus, IV drug use, spinal surgery/ procedures, infection	Avoid LP Blood cultures (60–70% sensitive) ESR < 20 mm/hour rules out SEA (98% specificity) CT myelogram, MRI	IV antibiotics, surgical evacuation
Spinal epidural haematoma	Spontaneous (rare), associated with thrombocytopaenia, bleeding diatheses, anticoagulation, vascular anomaly	Most often post-procedural (lumbar puncture, epidural, etc.)	Platelet count, coagulation studies; non-contrast CT	Surgical evacuation, Minor, stable deficits can be observed (often have complete recovery)
Granuloma	Most common TB (Pott's disease); also includes sarcoidosis	TB; spinal disease may worsen after initiation of TB therapy	CXR, spinal XR, PPD, sputum AFB, interferon assay; LP with AFB smear/ culture	TB: anti-TB regimen ± corticosteroids Sarcoidosis: corticosteroids, immunomodulatory agents
Schistosomiasis	Rare cause, CNS egg deposition; *S. mansoni* and *S. haematobium* most common	Subacute onset Lumbar predominance	Stool samples for ova, CBC with eosinophilia	Surgical decompression antihelminthics (1–3 doses) long-term corticosteroids

177 Transient ischaemic attack

TIA is defined by the American Stroke Association as *a transient episode of neurologic dysfunction caused by focal brain, spinal cord, or retinal ischemia, without acute infarction.* The prior time-dependent condition of recovery within 24 hours no longer defines TIA, as many patients with true infarction may also recover function within 24 hours. (Prior research showed that up to a third of patients diagnosed with TIA actually had MRI evidence of acute stroke.) This new definition, however, depends on the availability of advanced neuroimaging, and alternate proposals include ‘clinical symptoms typically lasting less than one hour, and without evidence of acute infarction’, where the evidence may include clinical or imaging findings. Some sources favour the term ‘acute neurovascular syndrome’ when imaging is not available. WHO still includes the condition of a clinical syndrome lasting less than 24 hours in many documents.

Risk factors for TIA and ischaemic stroke are the same (📖 p. 464), and many patients who present with TIA will have a stroke in the near future (up to 30% within three months in some settings).

The first five minutes

- ABC, VS, O_2, IV, blood glucose; monitor
- Rapid neurologic assessment
- Identify time of onset and duration of symptoms

History and physical examination

See 📖 p. 464 Carotid Doppler studies may show atherosclerotic for essential history and physical examination.

Risk factors and risk assessment

Every patient presenting with TIA should have an aggressive evaluation looking for modifiable risk factors.

- History may identify vascular risk factors, including hypertension, diabetes, hypercholesterolaemia, renal

disease, smoking, etc.
- Physical examination may identify dysrhythmias, CCF, carotid bruits
- ECG may show arrhythmias, especially AF, and evidence of hypertensive heart disease
- Echocardiography may show AF, evidence of hypertensive heart disease, dilated atria, clot, abnormal valve, or other potential thromboembolic source
- Carotid Doppler studies may show atherosclerotic plaque, a potential thromboembolic source

All patients should be risk stratified. One possible tool is the ABCD2 score (although there is an overall lack of regionally-relevant evidence). The higher the score, the higher the patient’s subsequent risk of stroke:

A: Age > 60 years	1 point
B: SBP > 140 mmHg, DBP > 100 mmHg	1 point
C: Clinical features: • Unilateral weakness • Abnormal speech; no weakness	2 points 1 point
D: Duration: • Greater than 1 hour • Less than 1 hour	2 points 1 point
D: Diabetes	1 point

Differential diagnosis

Includes seizure as well as vascular, infectious, oncologic, and other causes (📖 Stroke, p. 464).

Management and disposition

The goal of acute management is estimation of stroke risk, and identification and modification of ischaemic risk factors to minimise risk of stroke.
- All patients should undergo risk stratification and have a plan to modify risk
- Modification of risk factors should be managed with inpatient and/or primary providers and usually involves starting antiplatelet and statin therapy, smoking cessation, as well as treatment or prophylaxis for other risk factors (e.g. anticoagulation for atrial fibrillation)
- Admit those at high risk, those with risk factors that require inpatient treatment, and those who do not have access to outpatient evaluation within 24–48 hours; consider admission for young patients and those with multiple TIAs

Critical documentation

Document full neurologic examination, risk stratification and investigations performed and booked.

178 Acute stroke

One of the leading causes of admissions and death in urban African hospitals, stroke is a sudden loss of neurological function due to interruption of blood supply to the brain. There are two major types: ischaemic and haemorrhagic. Ischaemia may be thrombotic or embolic; intracranial haemorrhage may be intracerebral or subarachnoid (📖 Subarachnoid haemorrhage, p. 468).

Important risk factors for ischaemic stroke include hypertension, DM, hypercholesterolaemia, renal disease, smoking, hypercoagulable states, structural heart disease and AF. Both HIV infection and therapy may be associated with increased risk of ischaemic vascular events. Haemorrhagic stroke may be associated with hypertension, bleeding disorders, CNS infection (e.g. herpes encephalitis), septic emboli, vasculitis and vascular malformation. Stroke syndromes may also result from underlying lesions including tumours, toxoplasmosis, brain abscess, and other space-occupying lesions. Because there are overlapping risk factors and clinical features, distinguishing between haemorrhagic and ischaemic stroke in settings without neuroimaging can be challenging.

The first five minutes

• ABC, VS, O_2, IV access, cardiac monitor
• Check glucose
• If you have an institutional thrombolysis protocol, is patient eligible?

History and physical examination

Key historical features

Time of onset (and time when last seemed normal). Any progression or waxing and waning of symptoms? Was patient responsive throughout? Any tonic-clonic movements? Any recent change in activity or prior episodes of weakness, speech problems, confusion or clumsiness? Other vascular conditions or risk factors (cardiac or renal problems, diabetes, etc.). Any recent illness, trauma, or surgery? Any history of bleeding/clotting problems? Any use of anti-clotting medications (aspirin, clopidogrel, warfarin) or sympathomimetics (cocaine, amphetamines, traditional therapies)? Any history of brain lesions or procedures?

Signs and symptoms

Look for AMS and focal neurological deficit.
• Cardiovascular: haemodynamic compromise, rhythm (AF?), murmurs, cardiomegaly, absent pulses, carotid bruit
• Neurological: complete exam, focusing on LoC, risk for airway compromise, focal deficits and any evidence of seizure activity (gaze deviation, repetitive movements). Always evaluate the ability to speak, swallow and walk and use specific tests for cerebellar deficits. Look for more subtle signs, such as sensory deficits and neglect syndromes
• *Motor loss*: weakness, may be partial (paresis) or total (paralysis), and may be focal or general; pronator drift is a test for subtle weakness
• *Sensory deficit*: decreased sensation; deficit may be complete, or limited to pain/temperature, light touch, etc., and may be unilateral or focal
• *Aphasia*: impaired ability to formulate or understand language (spoken or written)
• *Apraxia*: inability to execute familiar tasks despite intact understanding and strength
• *Visual*: loss of vision in particular field, hemianopia (patient often unaware until tested)
• *Neglect*: inattention to one region of surroundings (e.g. left-sided neglect)
• *Other symptoms*: altered consciousness, dysphagia, dysarthria, ataxia, diplopia, and quadriparesis

Assess using the stroke scale recommended in your facility (NIH stroke scale, Hunt and Hess Scale or ROSIER (Recognition of Stroke in the Emergency Room)).

Table 178.1 Vascular localisation of stroke syndromes

Artery	Main clinical findings
Internal carotid artery	Hemiplegia, (arm = face = leg) Hemisensory deficit Hemianopia
Anterior cerebral artery	Hemiplegia (leg > arm)
Middle cerebral artery	Hemiplegia and numbness (face = arm > leg) AND aphasia *(if dominant hemisphere involved), OR AND hemi-neglect (if non-dominant hemisphere involved) Hemianopia
Posterior cerebral artery	Hemianopia
Lacunar	Pure hemiplegia (face = arm = leg) Pure hemisensory (face = arm = leg) Ataxia Sensorimotor: weakness and numbness of face, arm, leg, with NO cortical signs (no aphasia or neglect)
Vertebro-basilar arteries (brain stem)	Dysphagia, dysarthria, hemiplegia/quadriplegia Cranial nerve palsies Ataxia

left hemisphere is dominant in > 90% right-handed and in ~ 70% left-handed persons

Differential diagnosis

Hypertensive encephalopathy, hypoglycaemia, complex migraine, vasculitis, carotid dissection, delirium, cerebral oedema, seizures, CNS infection, malignancy, SOL (e.g. tumours, toxoplasmosis, brain abscess).

Investigations

- Labs: glucose, LP for suspected bleed if no contraindication and CT not available ◇; coagulation studies ◈
- Imaging: CXR (aspiration, malignancy) ◇; non-contrast CT brain (for haemorrhage), MRI brain (for ischaemic stroke), echocardiography (as indicated) ◈

Management

The goal of acute management for all cases is recognition of stroke, correction of hypoglycaemia, and protection of the airway if required. Give nothing by mouth until swallowing evaluated. Treat fever with antipyretics. Elevate the head of the bed 30°.

Ischaemic stroke

Management of ischaemic stroke focuses on protecting the airway, maintaining cerebral perfusion, prevention and early recognition of progression or haemorrhagic conversion, and identification and (usually inpatient) modification of thromboembolic risk factors to prevent recurrence. Do not lower BP in ischaemic stroke unless other hypertensive emergency (e.g. aortic dissection, MI), then only ~15% over 24 hours.

Thrombolysis

This remains a highly contentious issue in ischaemic stroke care, with limited regionally-relevant literature to guide practice. The therapeutic risk/benefit ratio is highly dependent on the availability of neuroimaging, coagulation studies and ICU-level care and is controversial even in settings where all of these are available. There are many contraindications to thrombolysis and it should only be undertaken in strict accordance with institutional protocol and in consultation with inpatient services.

We do not recommend thrombolysis in stroke in most settings. However, if your unit undertakes the practice, early identification of candidates is essential. Note that trials using streptokinase were negative – only consider thrombolysis using tPA or rtPA.

Haemorrhagic stroke

Management of subarachnoid haemorrhage is discussed elsewhere (🕮 p. 468). Management of intracerebral haemorrhage focuses on preventing expansion of bleed and controlling ICP and BP to maintain cerebral perfusion.

- Discontinue all anticoagulant and antiplatelet drugs, and reverse anticoagulation
- BP management in spontaneous haemorrhagic stroke:
  - SBP > 200 mmHg or MAP > 150 mmHg: consider IV infusion to lower ~20%. See 🕮 p. 106.
  - SBP > 180 mmHg or MAP > 130 mmHg: consider intermittent IV or infusion therapy to target MAP of 110 mmHg or 160/90 mmHg
  - In patients with elevated ICP, the goal is cerebral perfusion pressure (CPP = MAP–ICP) of 60 to 80 mmHg
  - Useful IV agents include labetalol, nicardipine, esmolol, enalapril, hydralazine, nitroprusside, and nitroglycerin, but all require careful monitoring (at least q15 min BP and neurologic checks)

Critical documentation

Serial VS and neurologic examination; progression of symptoms and response to interventions; investigation results.

Disposition

Admit all new strokes for evaluation, education and rehabilitation.

179 Subarachnoid haemorrhage

Bleeding into the subarachnoid space may be traumatic or spontaneous (caused by rupture of aneurysm; usually congenital). The cardinal symptom is acute headache. Spontaneous SAH is rare before the third decade and carries a mortality rate of 30–50%. Risk factors include: family history of SAH, hypertension, smoking, alcohol and stimulant abuse, connective tissue diseases (such as Marfan syndrome) and female gender.

Prompt recognition and management are essential to prevent death and disability.

The first five minutes

ABC, VS, O_2, glucose, cardiac monitor

History and physical examination

Key historical features

Sudden severe headache that is maximal within minutes of onset (although the sensitivity and specificity are not high; sudden onset is more useful than severity). Often described as the worst headache ever. Patients may report a headache in the preceding days or weeks – the promontory headache.

Nausea, vomiting, syncope, confusion and diplopia. Ask about risk factors, particularly a family history of SAH.

Signs and symptoms

- Level of consciousness (able to protect airway?)
- Focal neurological deficits
- Seizures
- Features of meningism (neck stiffness)
- Hypertension
- Arrhythmias (acute AF associated with SAH)
- Features of neurogenic pulmonary oedema (dyspnoea, hypoxia, pulmonary crepitations)

Differential diagnosis

- CNS infections (meningitis, encephalitis, abscesses)
- Severe primary headaches (migraine, cluster headache)
- CNS tumours
- Stroke
- Hypertensive encephalopathy
- Cavernous sinus thrombosis
- Any other cause of headache and/or neurological abnormalities

Investigations

Most SAH are caused by ruptured aneurysms.

In 30–50% of cases, rupture is preceded by a small leak causing severe headache or seizure a few days to three weeks prior to rupture. The sensitivity of CT for these small bleeds is poor (as low as 60% for minor SAH) and LP is essential for suspicion of SAH with normal CT. If not contraindicated (📖 p. 826), LP can be used as the sole diagnostic test, as it has near 100% sensitivity when CSF analysed for blood and xanthochromia.

The critical investigations are: CT, LP, and glucose.

- Labs: CBC, electrolytes, HIV, malaria, LP (📖 p. 826; look for > 100 000 RBC/mm3 and xanthrochromia) ◇; PT/PTT ◈
- ECG ◇: peaked, deep or inverted T-waves; prolonged QT; large U-waves, arrhythmias
- Imaging: CT brain (early scanning has a higher yield; may provide alternative diagnosis), CT angiography (superior), MRI (superior at detecting sub-acute and chronic haemorrhage) ◈

Management

The goal of acute management is prompt recognition, physiological stabilisation, airway protection and rapid neurosurgical referral.

- Elevate head of bed 30°
- Discontinue or reverse anticoagulants and anti-platelet medications
- BP control: SBP < 160 mmHg or diastolic ≤ 100 mmHg or MAP to ≤ 110 mmHg – nicardipine, labetalol, nitroprusside or hydralazine
- Control seizures with benzodiazepines
- Prevent cerebral vasospasm with calcium channel blockers (nimodipine (orally or NGT) or nicardipine (IV))

Critical documentation

Onset, clinical findings, LP and CT results; referral.

Disposition

Admit all patients, ideally to neurosurgery⟐.

180 Approach to vertigo and dizziness

Vertigo is the abnormal perception of movement where no movement exists. Patients describe a sensation that they are moving relative to the environment or the environment is moving relative to them. Classically, it is a ‘spinning’ sensation. It should be distinguished from lightheadedness, pre-syncope or faintness. It arises from signals from the semicircular canals, via CN VIII to the brainstem; problems can occur anywhere along the pathway and vertigo can represent benign or very serious disease.

Peripheral vertigo:

- Disease in the inner ear or along the peripheral nerve
- Usually benign
- Most common cause of vertigo

Central vertigo:

- Disease within the brain, brainstem or associated vessels

Dizziness is a non-specific term that may be used by patients to indicate true vertigo, light-headedness, imbalance, or a form of syncope. Dizziness may be a presenting symptom in many diseases and conditions, including myocardial ischaemia, stroke, infection, anaemia. Maintain a broad differential diagnosis.

The first five minutes

- ABC, VS, IV, cardiac monitor
- ECG

History and physical examination

Key historical features

Obtain an accurate, unprompted description of the patient’s ‘dizziness’ to determine whether true vertigo, near syncope, LoC, or non-vertiginous dizziness. Time of onset; associated hearing loss or ringing in ears; fever; trauma or blood loss; difficulty walking; weakness, numbness, or associated stroke symptoms; psychiatric history; medications; vascular risk factors, including hypertension, hyperlipidemia, diabetes, age, and family history.

Signs and symptoms

See Table 180.1.

Table 180.1 Central vertigo versus peripheral vertigo

	Central vertigo	**Peripheral vertigo**

Location of lesion	Brainstem or cerebellar	Inner ear or vestibular system
Onset	Gradual	Sudden
Nausea and vomiting	Occasional, mild to moderate	Frequent, moderate to severe
Symptoms	• Dysarthria • Dysphagia • Diplopia • Difficulty ambulating • Risk factors for stroke	• Tinnitus, hearing loss • Pressure or fullness in ear • Severe vertigo precipitated with movement • History of similar episodes
Signs	• Non-fatiguing horizontal nystagmus • Vertical/up-beating nystagmus • Cranial nerve III, IV, VI palsies • Abnormal cerebellar testing: finger-to-nose, heel-to-shin, rapid alternating movements • Ataxic gait	• Fatiguing horizontal nystagmus (could still be central) • Patient lying very still, often holding an emesis basin • Positive Dix-Hallpike testing

Differential diagnosis

- Nystagmus: may also be caused by CNS drugs (including phenytoin, see 📖 Toxicology p. 674)
- Peripheral vertigo: vestibular neuritis, benign paroxysmal positional vertigo (BPPV), post-concussive labyrinthopathy, Meniere’s syndrome, migraine, autoimmune disease, ototoxic drugs, mastoiditis, acoustic neuroma
- Central vertigo: cerebellar stroke, basilar artery thrombosis

Investigations

Non-vertigo dizziness

Broad work-up is based on suspected aetiology. May include:

- Labs: CBC, electrolytes, renal, urinalysis, glucose, pregnancy test, malaria ◇; cardiac enzymes, D-dimer ◈
- ECG ◇ (ischaemia, dysrhythmia)
- Imaging: CXR ◇; CT head, echo ◈

Peripheral vertigo

Usually a clinical diagnosis; no investigations needed. Consider blood glucose, electrolytes if vomiting severe.

Central vertigo

Assess for stroke (📖 p. 464).

Management

The goal of acute management is reduction of symptoms, timely management of causes of central vertigo, and preventing associated complications.

Peripheral vertigo

- Patient education and reassurance
- Epley manoeuvre may relieve symptoms entirely and, if successful, should be taught to the patient and family for home use
- Acute episode should improve or resolve within 2–3 days
- Pharmacotherapy:
  - » Meclizine or similar motion sickness medications may be used for control of mild to moderate peripheral vertigo (25 mg PO Q8h hours as needed)
  - » Benzodiazepines are the mainstay in severe acute peripheral vertigo: diazepam (2–5 mg PO Q8h–12 hours as

needed)
» Antiemetics give additional nausea and vomiting control: promethazine (25–50 mg PO Q6h) OR odansetron (4–8 mg PO Q8h) as needed

Central vertigo

See also 📖 Stroke, p. 464.
• Symptom control with anti-emetics (as above)
• Basilar artery thrombus is a neurology emergency; may require anticoagulation

Critical documentation

Serial neurological examination; distinguishing features of central versus peripheral vertigo; investigation results; response to acute therapy.

Disposition

Admit patients with peripheral vertigo and intractable vomiting, signs of infection or severe dehydration, or inability to take oral fluids or to ambulate. Refer central and unclassified vertigo to neurology (where available).

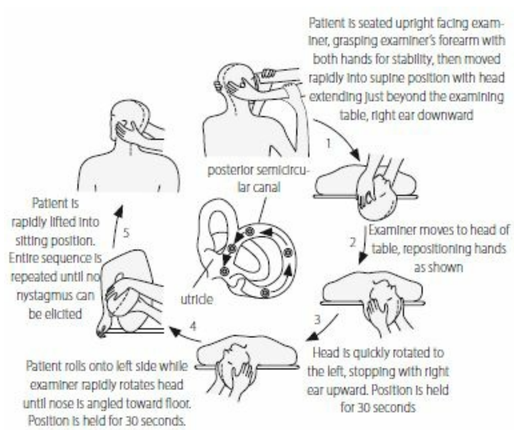


Figure 180.1

Dix-Hallpike manoeuvre

If semicircular canal canaliths are present, Dix-Hallpike manoeuvre will provoke vertigo and nystagmus (usually horizontal; appears a few seconds after movement; recedes in 30 s). (A). Start with patient sitting and head turned 45° to one side. (B). Place patient supine quickly with head over edge of bed. Observe for nystagmus for 30 s. (C). Sit patient up and observe for nystagmus for 30 sec. Repeat for other side.

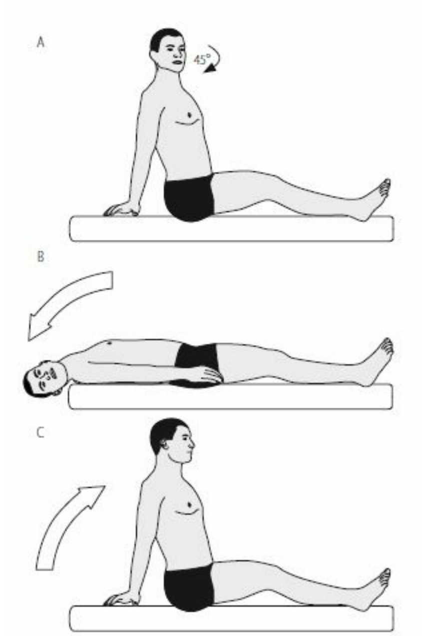


Figure 180.2

Epley's Manoeuvre

Start with patient sitting up with head turned 45° to affected side. Maintaining head's angle, have patient lie back with head tilted back off the end of the bed. Wait 30–60 s. Turn head 90° until at a 45° angle toward the unaffected side. Roll patient onto unaffected shoulder. Hold this position 30–60 s. Swing legs over the side of the bed and sit up. Keep chin tucked down 30–60 s. Repeat as necessary if symptoms recur.

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Part

4

K. Obstetrics and gynaecology

181 Abnormal uterine bleeding

Abnormal uterine bleeding (AUB) is a common acute complaint. In nonpregnant patients, most cases are not life threatening and can be managed as outpatients. AUB is any bleeding that occurs before menarche or after the menopause, or bleeding that occurs out of the normal menstrual cycle (regardless of duration, amount or interval).

The first five minutes

• ABC, VS, IVF – as needed (check for haemodynamic instability)

Possible causes and differential diagnosis

1. **Pre-menarche**: trauma, sexual abuse, foreign body and infection.
2. **Pre-menopausal**: bleeding that is out of normal menstrual cycle (normal menstrual cycle = 21–35 days, duration 1–7 days; average bleeding = 50 ml/cycle). Types: **menorrhagia** (prolonged/excessive but regular bleeding), **metorrhagia** (bleeding between menstruations), polymenorrhoea (regular, interval < 21 days), **oligomenorrhoea** (interval > 35 days), **anovulatory** (irregular, unpredictable). Causes include: pregnancy, infection/inflammation of the endometrium, cervical cancer, clotting disorders, medical illnesses (liver disease, polycystic ovarian syndrome, hormonal imbalance).
3. **Post-menopausal**: cervical cancer, endometrial cancer, uterine infection, fibroids/polyps, atrophy of the vaginal or uterine tissue, systemic diseases.

History and physical examination

Key historical features

Ask about possible pregnancy; the last menstrual period – frequency, amount and pattern of bleeding; post-coital

bleeding; frequent bruising; pelvic pain.

Ask about endocrine symptoms: sweating, weight loss or gain, palpitations, galactorrhoea.

Ask about any history of hepatitis (risk of coagulopathy) or HIV (risk of cervical cancer), history of trauma or sexual assault.

Signs and symptoms

Evaluate the patient: look for generalised signs of anaemia, or possible signs of an endocrine syndrome such as obesity, acne and hirsuitism, and any associated features (thyromegaly or thyroid tenderness, signs of liver failure).

Check for a surgical abdomen (guarding or rebound tenderness), evaluate for excessive amount of blood in the vaginal vault, you should be able to clear clots without recurrence. Do a bimanual exam to evaluate for uterine/cervical masses.

Investigations

- Labs: CBC (anaemia), electrolytes, LFTs, type and cross, pregnancy test ◇; PT/PTT ◈
- Imaging: US (fibroids, ectopic pregnancy) ◇; bedside US (free fluid and possible haemorrhage) ◈
- PAP smear: as an outpatient to screen for cervical cancer

Endometrial biopsy: as outpatient, especially for women >35 years (or <35 years with risk factors for endometrial cancer).

Management

The goal of acute management is to stabilise the patient and control bleeding if severe. Consider transfusion as needed.

If trauma on exam

- Repair any lacerations to the perineum/vagina
- Ask about sexual assault (p. 504)

If HCG positive

- Assess dates by last menstrual period and uterine size
- See First trimester bleeding p. 480 and Antepartum haemorrhage p. 486

If HCG negative and severe acute bleeding

- If actively bleeding: start oral contraceptive pill (OCP) and oestrogen 25 mg IV Q4h 1 day ◈ (and antiemetic as needed). Start iron replacement
- If stable: oestrogen 2.5 mg PO QID hours ◇. Start iron replacement
  - D&C if no response after 2–4 doses

If HCG negative and no severe acute bleeding

- Initiate OCP if no secondary cause suspected (consider pelvic inflammatory disease if CMT); NSAIDs can treat pain and reduce bleeding

If post-menopausal

- Stabilise; consider work up for malignancy

Critical documentation

Document repeated VS, amount of blood loss, US results and response to therapy

Disposition

Admit all patients with significant blood loss or continued bleeding or haemodynamic instability.

Discharge and follow up patients with no severe blood loss with haemodynamic stability and controlled bleeding.

182 First trimester vaginal bleeding

Bleeding in early pregnancy is common. Women of child-bearing age with vaginal bleeding should always be presumed pregnant.

The first five minutes

- ABC, VS, IVF – as needed. Pregnancy test.

History and physical examination

Key historical features

- Ask about: LMP, current pregnancy progression, prenatal care; degree and duration of bleeding; associated symptoms (abdominal pain, cramps, fever, dizziness, syncope). Risk factors for an ectopic pregnancy: tubal surgery, PID, prior abortion, IUD

Signs and symptoms

Evaluate the patient for signs of haemodynamic instability and pallor.

Detailed examination of the abdomen and pelvis:

- Abdominal tenderness and gestational age
- Pelvic examination: is the internal cervical os open or closed, are there clots or products of conception (POC) in the vaginal vault, adnexal mass, adnexal or uterine tenderness, lacerations or tears?

Possible causes and differential diagnosis

- Ectopic pregnancy: gestational sac outside uterus. Peritonitis or abnormal VS suggest rupture (life threatening). Always rule out. Unlikely if IUP seen on US (heterotopic pregnancy (both IUP and ectopic) 1:4 000)
- Recent induced abortion: or complications
- Spontaneous abortion: may be inevitable (bleeding with open internal os), incomplete (open os, POC visualised), complete (closed os, fetus and placental materials fully expelled)
- Septic abortion: fever and abdominal pain; can complicate any abortion
- Threatened abortion: bleeding and closed internal os; US shows IUP. Risk of completed abortion 35–50%
- Molar pregnancy: abnormal trophoblastic tissue. Bleeding at 12–16 weeks, uterus larger than expected, passage of grape-like material, US with ‘snowstorm’ pattern
- Non-pregnancy-related vaginal or cervical pathology

Investigations

Always examine passed uterine contents: rinse and place in saline/tap water. Blood dissolves, chorionic villi will be fluffy and fingerlike – presence of villi excludes ectopic.

- Labs: Hgb/hct, type and cross, Rh, pregnancy test ◇
- Imaging: pelvic US (transvaginal or transabdominal) ◇:
  - » IUP: intrauterine double gestational sac, fetal pole or heart activity
  - » Ectopic: pregnancy outside uterus, ectopic fetal pole or heart activity
  - » Suggestive of ectopic: moderate free fluid or adnexal mass without IUP
  - » Indeterminate: empty uterus, nonspecific fluid, single gestational sac/pseudosac. IVP unlikely if no gestational sac on transabdominal US with hCG > 6 500 or transvaginal US with hCG > 3 000 (6–7 weeks)
- Culdocentesis: if US not available (detects hemoperitoneum, but less sensitive and specific; if positive, treat as ruptured ectopic)

Management

The goal of acute management is fluid resuscitation, and urgent surgical intervention if ruptured. IVF and blood as needed; anti-D immunoglobulin if Rh-negative (50 µg in first trimester, 300 µg after first trimester), analgesia.

Ectopic

Consult obstetrics early.

- Laparotomy ◇: unstable, peritoneal signs, evidence of rupture
- Methotrexate ◈ (50 mg/m^2 BSA IM × 1) only if definite ectopic by US, tubal mass < 3.5 cm, no fetal cardiac activity, no evidence of rupture, minimal pain, hCG < 5 000. Needs close follow-up
- If US indeterminate or not available: admit for serial exams. Or, if patient reliable, pain free, stable, pregnancy < 6 weeks, and resources available, discharge for repeat hCG in 48 hours and US in 7 days

Spontaneous abortion

- If heavy bleeding, transfuse as needed
- Gently remove fetal tissue if visualised in cervical os
- If febrile, doxycycline 100 mg, ceftriaxone 1 gm IV and metronidazole 500 mg IV) OR (ampicillin 2 gm IV, gentamicin 2 mg/kg IV and metronidazole 500 mg IV)
- If prolonged bleeding, unstable, or febrile, needs surgery

Threatened abortion

Bleeding with live IUP on US, closed os.

- Expectant management; close follow-up with OB

Molar pregnancy

- Consult OB, D&C, ensure OB follow-up to trend hCG

Disposition

Admit for ruptured ectopic, haemodynamic instability, falling haematocrit, severe pain, fever. Discharge if abortion with minimal blood loss and good OB follow up. Return if fever, worsening abdominal pain, significant increase in bleeding.

183 Hypertensive disorders during pregnancy

Hypertension in pregnancy is rarely benign and may be indicative of life threatening eclampsia/pre-eclampsia.

Definition

Hypertension (HTN)

SBP ≥ 140 mmHg or diastolic blood pressure (DBP) ≥ 90 mmHg, on two occasions six hours to seven days apart; or one BP ≥ 160/110 mmHg.

Proteinuria

24-hour urine specimen > 0.3 g protein (or ≥ 1+ on dipstick OR ≥ 100 mg/l on two random urine samples collected at least four hours apart).

The first five minutes

- ABC, VS, IV access, monitors – as needed
- If there is evidence of eclampsia (HTN associated with coma/seizures) mobilise staff, start $MgSO_4$

History and physical examination

Key historical features

Ask about: pregnancy, pre-natal care, medication; associated features (bruising and bleeding, frothy urine); history of HTN and complications in previous pregnancies.

Signs and symptoms

- Evaluate the patient for signs of AMS, generalised oedema, bruising
- Detailed examination of the abdomen and pelvic examination, assess gestational age

Differential diagnosis

- Chronic HTN: HTN antedates pregnancy. Consider preeclampsia/eclampsia if:
  - » New proteinuria after 20 weeks GA, or sudden increase in pre-existing proteinuria
  - » An exacerbation of BP to the severe range, or severe symptoms
- Gestational HTN: HTN during pregnancy, without proteinuria or other signs of preeclampsia
- Preeclampsia: HTN + proteinuria after 20 weeks. Mild or severe. Severe if:
  - » BP ≥ 160/110 mmHg or urine protein ≥ +3 (or > 5 gm/24 hr urine)
  - » Severe symptoms: headache, visual changes, epigastric pain, severe oedema, IUGR, abruptio placenta, oligouria, DIC (bleeding, petechial), HELLP syndrome (haemolysis, elevated liver enzymes and low platelets), vomiting
  - » Platelets < 100 000, altered liver and renal function tests
- Eclampsia: grand mal seizures or coma in a woman with preeclampsia

Investigation

Helps in diagnosis and determining severity of the disease.

- Labs: CBC, renal, type and crossmatch, urinalysis ◇; urine protein, LFTs, platelet count, PT/PTT ◈
- Imaging: US ◇
- Fetal non-stress test ◇

Management

The goal of acute management is to control blood pressure and find and treat complications.

Gestational hypertension

Check blood pressure and protein. Refer for outpatient management. If BP remains in mild range, proceed with normal labour and childbirth at term.

Eclampsia and pre-eclampsia

	Severe pre-eclampsia	**Eclampsia**
When to deliver	If mild, deliver at 37 weeks (refer for outpatient follow-up) If severe, 34 weeks or end organ damage: deliver immediately. If < 34 weeks: give steroids, observe as inpatient, deliver at 34 weeks	Immediately (< 12 hours) deliver by C-section if vaginal delivery not possible in this time Transfer patient as needed
Initial measures	ABC	ABC; O2; IV antibiotics
When to give seizure prophylaxis	During evaluation, labour and continued for 24 hours after delivery	Immediately; continue until 24 hours after delivery or last seizure
General measures when admitted	Monitor urine output (goal > 0.5 ml/kg/hr, fluid balance; VS; fetal HR; reflexes and signs of pulmonary oedema.	
Acute blood pressure control	1st line: hydralazine: 5–10 mg IV every 5 minutes until DBP < 110 mmHg. Repeat hourly as	

(goal DBP 90–100 mmHg and SBP 140–150 mmHg)	needed (or 12.5 mg IM every 2 hours) 2nd line: nifedipine: 5–10 mg sublingually, then 5–10 mg in 30 minutes if response inadequate. Then 10–20 mg PO every 6 hours (10–40 mg PO bid maintenance). 2nd line: labetolol: 20 mg IV push over 2 minutes. Repeat as needed every 10 minutes, doubling the dose up to 80 mg for desired effect. Maximum total cumulative daily dose is 300 mg IV.
Seizure prophylaxis: Note: magnesium ($MgSO_4$) is critical to stopping and preventing convulsions	**Magnesium**: before administration: ensure RR > 16/minute, reflexes are present Loading dose: $MgSO_4$ (20% solution) 4 g IV over 5 minutes. Then 10 g 50% MgSO4 IM, 5 g in each buttock. If convulsions recur after 15 minutes, give 2 g MgSO4 (50% solution) IV over 5 minutes Maintenance: 5 g MgSO4 (50% solution) + 1 ml lignocaine 2% IM every 4 hours into alternate buttocks If respiratory arrest: assist ventilation, give calcium gluconate 1 g (10 ml 10% solution) IV slowly until respiration begins **Diazepam**: second line. Risk fetal or maternal respiratory depression Loading dose: 10 mg IV over 2 minutes, if convulsions recur, repeat loading dose and 40 mg in 500 ml IV fluids; drops titrated to keep patient sedated but arousable Can give rectally when IV access is difficult: loading dose of 20 mg followed by maintenance dose of 10 mg/hr

Disposition

Admit all patients with eclampia or pre-eclampsia. Discharge patients with HTN or gestational HTN and manage as out-patients (if follow-up permits).

184 Obstructed labour and ruptured uterus

Obstructed labour (OL) is failure of descent of the fetus in the birth canal for mechanical reasons, in spite of good uterine contractions. OL is a neglected labour and should not occur in a labour ward. It is an emergency and requires a concerted team approach.

Causes

- Cephalopelvic disproportion: small/abnormal pelvis or large fetus
- Congenital fetal abnormalities, locked twins, shoulder dystocia, fetal malpresentations and positions, or abnormal reproductive tract

Diagnosis

Obstructed labour

- History of labour for days, maternal exhaustion, anxiety, confusion, or unconsciousness, shock
- Abdominal examination:
  - » Fetal head above the pelvic brim, abnormal or no fetal heart tone
  - » Distended and tender abdomen, Bandl’s ring (a thick ring is felt as high as the umbilicus)

Ruptured uterus

Common in multiparous women.

- Exam: shock, haemoperitoneum, tender abdomen, easily palpable fetal parts
- Vaginal examination: foul smelling discharge, significant caput and moulding, oedema of the vulva

Complications

- Maternal: PPH, sepsis, urinary and rectal fistula, nerve injuries, uterine rupture
- Fetal/neonatal: fetal death, asphyxia, sepsis

Treatment

Simultaneously start resuscitation while identifying the cause and treating infection.
• Once OL is diagnosed, Caesarean is the rule. No place for instrumental vaginal delivery
• Destructive delivery (craniotomy) is an exception used only when the fetus is dead, there is no sign of uterine rupture or impending rupture, OL is in 2nd stage with engaged head, and a place with no operating room facility
• In case of ruptured uterus, emergency laparatomy

185 Antepartum haemorrhage (APH)

Antepartum haemorrhage, also known as pre-partum haemorrhage, is defined as vaginal bleeding during pregnancy from 24 weeks gestational age to term.

The first five minutes

• ABC, VS, IVF – as needed
• Assess for massive haemorrhage and hemodynamic instability

History and physical examination

Key historical features

Ask about
• Bleeding: duration, amount and antecedent event (trauma)
• The pregnancy: estimate gestational age, complications, pre-natal imaging
• Fetal movements

Signs and symptoms

• Evaluate the patient: look for signs of anaemia, obvious distress
• Detailed examination of the abdomen and pelvis: evaluate for rebound and guarding, estimate gestational age; perform an external pelvic exam to quantify the amount of visible bleeding or signs of imminent delivery such a fetal presenting parts. DO NOT perform a digital vaginal or speculum exam until placenta previa is ruled out

Investigations

• Labs: Hgb, type and group, Rhesus status ◇
• Imaging: US

Potential causes and management

• Placenta: abruptio placentae, placenta previa, vasa previa (rarely)
• Uterine: rupture
• Local: cervix, vagina and vulva. All local causes of APH have minimal spotting or bleeding. An exception to such a presentation is the occasional profuse bleeding of ruptured vaginal varicose vein. Once placenta previa is excluded, digital and speculum examination may confirm the specific local cause
• Preterm labour/bloody show
• Indeterminate: no cause identified even after delivery and examining the placenta
(See Table 185.1 for clinical findings).

Management

The goal of acute management is to stabilise the patient and ensure fetal wellbeing. In all cases a skilled obstetrician should guide management (if possible).
• In milder abruption and remote from term, use expectant management (admission, steroid administration) to prevent prematurity
• Moderate and severe abruption (irrespective of gestational age) and abruption at term (irrespective of degree of bleeding) need immediate delivery. A Caesarean section may be indicated for severe bleeding endangering

maternal life, fetal distress, when vaginal delivery seems unlikely within a reasonable time, and for other obstetric indications
• Identify and treat any coagulation defects or anaemia
• If mother is Rh negative administer 300 mcg rhogam

Table 185.1 Clinical findings in placenta previa and abruptio placenta

Clinical findings	Placenta previa	Abruptio placentae
Vaginal bleeding	Painless Recurring (often) Bright red	Painful Presence hypertension, trauma, etc. Non-recurring Menstrual like
Hypotension	In proportion to vaginal blood loss	Out of proportion to amount of vaginal bleeding
Uterus	Quiet or relaxed between labour contractions	Irritable, not relaxing between labour contractions (tetanic contraction)
Fetal presentation	Mal-presentation (transverse, breech), unengaged head	Difficult to palpate fetus Engaged head
Fetal condition	Usually normal	Fetal distress, fetal death, IUGR

Disposition

Admit as per specific condition.

186 Post-partum haemorrhage (PPH)

Defined as vaginal bleeding > 500 ml after a singleton vaginal delivery of > 28 weeks; if delivered by C-section or multiple vaginal birth, the definition changes to bleeding > 1 000 ml.

The first five minutes

ABC, VS, IVF – as needed. Assess for haemodynamic instability.

History and physical examination

Key historical features

Ask about the pregnancy.

Signs and symptoms

Evaluate the patient for signs of haemodynamic instability and pallor.

Detailed examination of the abdomen, pelvis and placenta will guide likely cause and management – see Table 186.1 for a comprehensive overview. Assess for estimated blood loss, uterine size and extent of contraction, and completeness of the placenta.

Investigations

• Labs: Hgb/haematocrit, type and cross, Rhesus, pregnancy test ◇

Management

The goal of acute management is acute resuscitation of the mother, and termination of bleeding.
• IVF and blood as needed; anti-D immunoglobulin if Rh-negative (300 μg)
• Consult obstetrics early

Disposition

Admit all patients; consider high care for those with haemodynamic instability, falling haematocrit, or uncontrollable bleeding.

Table 186.1 An overview of PPH

Cause	Causes	Features	Management
Atonic uterus (uterus not contracted)	Hypotonic uterus leads retention of the placenta and excessive bleeding	Commonest cause of primary PPH. Diagnose if: soft, not contracted uterus with fundus above the umbilicus	Stimulate contraction: massage the uterus. Oxytocine (20 IU/1 000 ml, 30 drops/min) No response: bimanual compression of the uterus; consider compression of the abdominal aorta
			Other uterotonics (misoprostol (800 mcg SL) or ergometrin (0.2 mg IM) Persistent bleeding: uterine tamponade (intrauterine balloon or condom tamponade (condom tied to end of foley catheter, inserted into uterus, and filled with 350 mm^3 NS)). If no response: uterine or utero-ovarian artery ligation, or hysterectomy
Retained placenta	Usually due to poor uterine contraction	Placenta may be retained without bleeding (pathological adherence)	Apply controlled cord traction (CCT) If CCT fails, manual removal of the placenta in OT; consider laparatomy
Trauma	Tears of the birth canal (including uterine rupture): feto-pelvic disproportion (obstructed labour), instrumental deliveries, scarred uterus.	Diagnosis: bright red (arterial) bleeding with a contracted uterus	If PPH with delivered placenta and well contracted uterus: Explore the genital tract manually and using speculum; repair vaginal/cervical tear; if uterine rupture detected laparatomy is indicated
Coagulation defects	Abruptio placentae, intrauterine fetal death, infection	See Coagulopathies p. 288	
Acute inversion of the uterus		The uterus may rarely turn inside-out during delivery. Causes shock by bleeding or neurogenic shock (increased vagal tone from stretching of the pelvic parasympathetic nerves). With the placenta detached, is described as cherry red mass	Under appropriate analgesia, apply: Immediate gentle upward transvaginal pressure. (Johnson technique – lift the uterus and the cervix into the abdominal cavity with the fingers in the fornix and the inverted uterine fundus on the palm; gently push the fundus back through the cervix. Keep hand in the uterus until the fundus begins to climb up). If the placenta is still attached, do not remove until the uterus is replaced through the cervix. Oxytocin only after successful replacement. If this fails, laparotomy

187 Management of labour and delivery

The first five minutes

Ask about uterine contractions, vaginal fluid or bleeding, fetal movement. Take IVS, check uterine contraction and fetal heart rate (FHR).

History and physical examination

Record all findings and interventions with exact time.

Key historical features

Gravidity, parity, gestational age in weeks, complications with this or prior pregnancy, time contraction started, frequency, fluid or bleeding; past medical illnesses.

Signs and symptoms

Detailed examination of the abdomen and pelvis: Leopold manoeuvre for fetal age and

presentation
Sterile digital vaginal examination (if no bleeding) to assess stage.

Investigations

If not already done, Hgb, blood group and Rh, urinalysis and microscopy, RVI.

First stage (labour onset to full cervical dilation 6–12 h)

Monitor maternal wellbeing

If patient in 1st stage or has high risk pregnancy (requiring obstetrician), immediately transfer to delivery unit. If unable to transfer or imminent delivery:

- IVS: every 30 minutes
- Emotional support, IV/IM opioids or epidural as desired

Monitor fetal wellbeing

- FHR: Pinnard stethoscope or continuous FHR monitor (◈) for 1 min after contraction; repeat Q30 minutes (low risk) or Q15 min (high risk)
- Monitor fluid for meconium: if moderate, likely fetal distress; if thick, definite distress.

Monitor progress of labour

- Contractions – frequency, duration and intensity (by palpation or toco-dynamometer); every hour (latent phase) or 30 min (active phase)
- Descent of fetal head: determine by abdominal palpation
- Vaginal examination (every four hours). Assess: cervical dilation, (normal progress 1 cm/hr), station, position, caput and moulding

Second stage (full cervical dilation to birth)

Normal duration: nullipara: < 2 hrs (3 hrs with epidural anaesthesia).
Multipara: < 1 hr (2 hrs with epidural). Longer is prolonged 2nd stage.

Monitor maternal wellbeing

- Evaluate: general condition, pain, hydration, VS every 30 minutes
- Empty bladder, avoid early pushing until head is visible

Monitor

- Mother: pain, VS every 30 min. Empty bladder. Avoid pushing until head visible
- Fetus: every 15 min (LR) or 5 min (HR)
- Progress: evaluate contraction every 10 min and descent every hour

Assistance of spontaneous delivery

- Episiotomy only if perineal resistance or if expedited delivery indicated
- Timing: when head distends vulva 2–3 cms. Types: median or mediolateral (mediolateral is preferable). Use local anaesthesia
- Assist delivery of head using modified Ritgen’s manoeuver if extension does not occur easily (hand protected with sterile towel placed on the perineum, fetal chin palpated and pressed upward gently effecting extension)
- Check for cord around neck: if present gently move over the head. If not reducible: deliver without reduction or clamp at two sites and cut in between

- After delivery of head: wipe mouth and oro-pharynx (routine suctioning not recommended). Allow for restitution of head. Then place a hand on each parietal eminence. Apply gentle downward pressure of the head toward the maternal sacrum to deliver anterior shoulder. Then deliver posterior shoulder by upward traction
- Put fetus at level of introitus for three minutes. Immediately dry the body. Clamp cord 4–5 cm from fetal umbilicus. Delayed clamping (after a few minutes) is associated with increased neonatal haematocrit. Take cord blood if indicated
- If second stage prolonged, manage the causes:
  - » Poor maternal pushing effort: consider vacuum or forcep delivery
  - » Poor uterine contraction: oxytocin
  - » CPD: Caesarean delivery

Third stage (birth to delivery of placenta and uterine retraction)

Lasts on 30 minutes ~Provide active management:

- Administer uterotonic agents immediately after fetal delivery (1st choice: oxytocin 10 IU IM; 2nd choice: misoprostol 600 mcg orally; 3rd choice: ergometrine 0.2 mg IM or syntometrine (1 ampoule) IM)
- Apply gentle, even cord traction to deliver placenta
- Uterine massage immediately after placental delivery

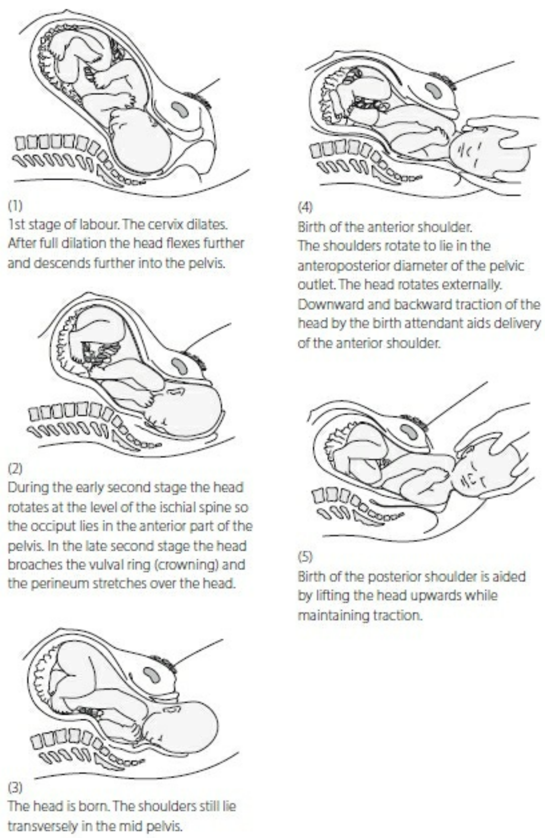

Figure 187.1 Stages of normal delivery

188 Breech presentation and delivery

A breech presentation can lead to death of both mother and child if not managed in a timely and efficient manner (see Figure 188.1 p. 495).

The first five minutes

- Confirm the presentation (use US if needed), fetal condition and presence of labour
- Start 2 large bore IVs, type and cross, call for obstetric assistance

History and physical examination

Key historical features

Gravity, parity, gestational age, problems with this or prior pregnancy.

Signs and symptoms

- Evaluate the patient for signs of hemodynamic instability
- Abdominal palpation (Leopold's manoeuvres), may reveal a round, hard, smooth mass (head) occupying the fundus and a soft, broad, indefinite and non ballotable mass (the breech) occupying the lower pole of the uterus
- On vaginal examination, three types of breech may be identified:
  - » Frank breech: buttocks in the pelvis, both legs extended
  - » Complete breech: one or both feet are felt alongside the buttock
  - » Footling breech: one or both feet are inferior to the buttock

Management

The goal of acute management is to ensure maternal then fetal well-being to delivery if imminent.

- If preterm and not in labour, refer to OB for expectant management (may turn)
- If GA > 37 weeks and if no contraindications, consider external cephalic version (ECV) (baby manipulated through abdominal wall into cephalic presentation). If ECV fails, consider breech vaginal delivery
- If in advanced labour do breech vaginal delivery. Caesarean only for footling breech (increased risk cord prolapse and arrest of head) or obstetric indications (APH, etc.)
- Induction or augmentation of labour is contraindicated

Types of vaginal breech delivery

- Spontaneous breech delivery: the baby is expelled entirely spontaneously
- Total breech extraction: deliver by extracting the entire body of the fetus from the uterus; rarely indicated except to expedite the emergency delivery of a second twin
- Assisted vaginal breech delivery: partial breech extraction if no cord prolapsed or entanglement
- Delivery of the breech, abdomen and shoulder:
  - » Keep in lithotomy position and empty bladder. Pudendal block and local infiltration; episiotomy when the fetal anus is visible. Instruct the mother to bear with contraction; allow the buttocks to deliver spontaneously; no other manipulation until delivered up to umbilicus
  - » If legs are not delivered spontaneously, assist delivery of one leg at a time by lateral rotation of thighs and flexion of knees using a finger
  - » Hold the baby by the hips (not by the flanks or abdomen as this may cause organ damage) with towel, fingers over anterior superior iliac spine and the sacrum. Apply gentle, steady downward traction with good maternal pushing until the lower half of the scapula is delivered
  - » Allow the arms to disengage spontaneously one by one; only assist if necessary. After spontaneous delivery of the first arm, lift the buttock towards the mother's abdomen to enable the second arm to deliver spontaneously. If the arm is not spontaneously delivered, place one or two fingers in the elbow and bend the arm, bringing the hand over the baby's face
  - » If arms are stretched above the head or folded around the neck, use Lovset's manoeuvre: hold the baby by the hips and turn half circle, keeping the back uppermost and applying downward traction so that the arm that was posterior becomes anterior and can be delivered under the pubic arch. Assist delivery of the arm by placing one or two fingers on the upper part. Draw the arm down over the chest as the elbow is flexed. To deliver the second arm turn the baby back half a circle (reverse direction) and follow the same procedure as for the first

arm

» If the baby's body cannot be turned to deliver the arm that is anterior, first deliver the shoulder that is posterior by holding and lifting the baby up by the ankles, move the baby's chest towards the woman's inner leg. The shoulder that is posterior will deliver then deliver the arm. To deliver the anterior shoulder, lay the baby back down by the ankles (depressing the body)

• Delivery of the head:

» Mauriceau Smellie Veit Manoeuvre (MSV): lay the baby face down with the length of its body over your hand and arm, with the middle and forefingers of your hand on the baby's cheekbones. Pull down to flex the head while the other hand grasps the baby's shoulders. With two fingers and middle finger placed and pushing over the suboccipit (hooking round the neck), gently flex the baby's face towards the chest, while applying downward pressure on the shoulder. Pull gently downward until the hairline is visible while an assistant applies suprapubic pressure. Once the head gets into the pelvis, following the pelvic curve, raise the baby until the mouth and nose are delivered

» Forceps delivery if MSV fails

Figure 188.1 Breech presentation

189 Shoulder dystocia

Shoulder dystocia is inability to deliver the shoulders after the fetal head has been delivered, despite the performance of routine obstetric manoeuvres. It is an obstetric emergency requiring skilful management to avoid significant fetal damage and death.

Higher risk of occurrence during delivery of > 4 kg babies, but shoulder dystocia may not be predicted. Be prepared for it at all deliveries, especially if a large baby is anticipated and in women with diabetes mellitus, previous history of large babies, and obesity. Asphyxia, birth injuries, injury to the brachial plexus and PPH are some of the complications.

Diagnosis

• The fetal head is delivered but remains tightly applied to the vulva

• The chin retracts and depresses the perineum. Traction on the head fails to deliver the shoulder, which is caught behind the symphysis pubis.

Management

Episiotomy should be performed at any point if needed to create adequate space for manoeuvres.

• In the lithotomy position, ask the woman to open and flex both thighs, bringing her knees as far up as possible towards her chest. Ask two assistants to push her flexed knees firmly up onto her chest (McRobert's manoeuvre, see Figure 189.1)

• Apply firm (not excessive), continuous traction downwards (towards the floor) on the fetal head while an assistant simultaneously applies suprapubic pressure downwards to assist delivery of the anterior shoulder

- In patients with only local or pudendal anaesthesia, an effective initial manoeuver may be to rotate the woman to a position of hands and knees and attempt delivery in this manner (Gaskin all-fours manoeuvre)

If not successful, continue with manoeuvres below:

- Insert a hand into the vagina and apply pressure on the back surface of the anterior shoulder in the direction of the baby's sternum to rotate the shoulder and decrease the width of the shoulders; if needed, apply pressure to the back of the posterior shoulder in the direction of the sternum (Rubin's manoeuvre).
- Alternatively, apply pressure to the anterior surface of the posterior shoulder until the baby turns and the anterior shoulder emerges from underneath the pubic symphysis. (Woods' screw manoeuvre)
- If the shoulder still is not delivered despite the above measures, grasp the humerus of the posterior arm. Keeping the arm flexed at the elbow, sweep the arm across the chest. This will provide room for the shoulder that is - anterior to move under the symphysis pubis
- If the posterior arm cannot be reached, apply traction with a hook in the axilla to extract the shoulder that is posterior; this may bring the posterior arm down sufficiently to be grasped and delivered

If all of the above measures fail, the last option is to fracture the clavicle (will decrease the width of the shoulders and free the anterior shoulder); apply traction with a hook in the axilla to extract the arm that is posterior (replacing the head and doing Caesarean is rarely successful). After this is done, repeat above manoeuvres with the newly shortened biacromial diameter.

Figure 189.1 McRobert's manoeuvre

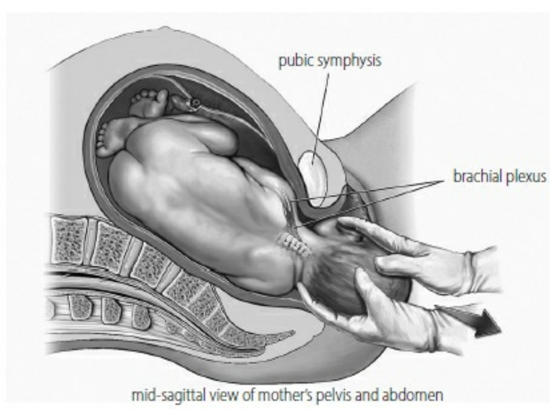

Figure 189.2 Shoulder dystocia

190 Caesarean section

Caesarean section is the delivery of the fetus(es), placenta and membranes through an incision of the abdominal and uterine wall at > 28 weeks of gestation. It can be elective or emergency.

Uterine incision either lower (most common) or upper segment. Indications can be obstetric or non-obstetric. Anaesthesia usually regional (spinal/epidural); general or occasionally used.

Procedure

- Skin incision. Pfannensteil, or sub-umbilical midline (for fast delivery)
- Make a small incision over the fascia with a scalpel; extend to the whole length of fascia with scissors
- Dissect the rectus and pyramidalis muscles by sharp instrument followed by blunt dissection
- Elevate the peritoneum at the upper edge of the incision by holding it with two artery forceps about 2 cm apart. Palpate the tent of peritoneum to check for omentum or bowel. If present, release the artery forceps and grasp again
- Incise between the two artery forceps with scalpel to open the peritoneal cavity. Check for adhesion of the peritoneum or dense infiltration by inserting a finger and palpating up and down the peritoneal opening. Extend the peritoneal opening with scissors up to the upper border of the incision and downward to the reflection of the bladder checking for adhesions
- Correct the uterus if dextro-rotated
- Insert moistened packs on each side of uterus and insert bladder retractor
- Lower segment transverse cesarean section:
  - » Grasp the peritoneal flap at the site of the reflection with forceps and incise with scissors. Dissect the peritoneal flap at the reflection site by inserting the scissors between the serosa and myometrium. Open up the instrument to dissect the peritoneum and then cut moving to the left and right side of the uterus (assistant moves the bladder retractor to the side you are moving the scissors). Push the peritoneum downwards with gauze on a holder or using your fingers
  - » Incise transversely over the exposed uterine lower segment for about 2 cm with a scalpel. The incision should be just enough to cut through the myometrium but should not reach the fetal parts. Extend the incision bluntly with your index fingers of the two hands laterally and upwards. Rupture the amniotic membrane if encountered
  - » Remove the bladder retractor. Then insert your right hand between the symphysis pubis and the presenting part and elevate the head (in case of vertex, brow or face presentation) gently through the incision assisted by gentle abdominal pressure
- Wipe the nares and mouth once the head is delivered. Deliver the rest of the body
- Anaesthetist administers oxytocine 10 IU IM (or 20 IU in 1 000ml NSV)
- Clamp the cord at two sites and cut in between. Hand over the neonate to the midwife for immediate newborn care
- Give one dose broad spectrum antibiotic (ampicillin 2 gm IV or a first-generation cephalosporin) IV immediately after the cord is clamped
- Deliver the placenta by cord traction. Clean the uterine cavity with pack to ensure completeness of the placenta and membranes
- Clamp the edges of the uterine incision and any briskly bleeding sites with green armitage or ring forceps. Lift the uterus out of the abdominal cavity and cover the fundus with moist pack. Close the uterine incision with two layers of continuous inverting stitches starting from the edge the first bite just behind the edge with Chromic 1- or 0-catgut or polyglycolic (Vicryl). Place the uterus back into the abdominal cavity. Make sure haemostasis is secured and uterus is well contracted. Dry the abdominal cavity with gauze pack if there is grossly contaminated amniotic fluid or meconium
- Close the fascia with continuous Vicryl no 2, approximate the subcutaneous layer with chromic 2-0 catgut, close the skin with continuous subcuticular stitch or interrupted silk as needed. Ensure uterine contraction and clean any clot in the vagina

191 Vaginal discharge

Vaginal discharge is a common complaint; most causes are benign if treated correctly.

The first five minutes

- ABC, VS, IV access – as needed
- Not usually life threatening. Consider pelvic inflammatory disease (PID) and ectopic pregnancy if shocked

History and physical examination

Key historical features

Ask about

- Abdominal pain, itching, and dysuria
- The last menstrual period
- Sexual history, oral contraceptive use, bleeding with intercourse
- Risk factors for infection; recent antibiotic use (*Candida*)

Signs and symptoms

Evaluate the patient for haemodynamic instability and febrile illness.

- Abdominal pain tenderness, signs of guarding or rebound tenderness
- Pelvic exam assess for cervical motion tenderness (CMT), ulcerations or lesions on the genitalia, discharge on speculum exam (physiologic discharge: thin, clear or white, without foul odour, pain, or pruritis)
- In children: examine external genitalia. Check for signs of abuse or foreign body. If the hymen is still intact avoid a pelvic exam

Possible causes and differential diagnosis

Infection

Bacterial vaginosis (overgrowth of vaginal bacteria); candidiasis (overgrowth of yeast); trichomoniasis (sexually transmitted protozoal infection); cervicitis or PID (often due to gonorrhoea or chlamydia).

Other

Retained foreign body; atrophic vaginitis; allergic or contact dermatitis; chemical irritation; severe condylomata acuminate; malignancy.

Children

Above causes, or nonspecific vulvovaginitis, pinworm, enteric flora, or foreign body; sexually transmitted diseases may occur due to sexual abuse.

Investigations

Investigations are directed towards the suspected cause.

- Labs: pregnancy test, urinalysis (if dysuria or frequency), vaginal pH (apply pH stick to vaginal side wall (not posterior secretions), pH of normal secretions in menstruating females 4–4.5), microscopic analysis of discharge, KOH whiff test (apply KOH to discharge; fishy odour = bacterial vaginosis) ◇; vaginal culture, PCR (*Gonococcus*, *Chlamydia* (do not delay treatment for results)) ◈

Management

Table 191.1 Management of vaginal discharge

Condition	Clinical features	Management
PID	Patients may be febrile and tachycardic, or have normal VS. May have vomiting or pain. Adenexal or uterine tenderness	IVF as needed Antibiotics: ceftriaxone 250 mg IM stat (for gonorrhoea) AND doxycycline 100 mg PO BID × 14 days (for *Chlamydia*) AND metronidazole 500 mg TID × 14 days (for anaerobes)
Cervicitis	Purulent cervical discharge, friable cervix on exam, bleeding of cervix with intercourse or exam	Treat for both gonorrhoea and *Chlamydia*: • Gonorrhoea: ceftriaxone 250 mg IM stat • Chlamydia: doxycycline 100 mg PO BID × 14 days

Candidiasis	Vaginal itching + discharge (white, curd like) without abdominal pain; *budding yeast or pseudohyphae* on microscopy	Fluconazole 150 mg PO stat OR topical and vaginal suppository of antifungal (e.g. nystatin or miconazole) × 3–7 days • Follow up if symptoms do not resolve • HIV testing if recurrent
Bacterial vaginosis and trichaomoniasis	Non-purulent discharge without pain; pH > 4.5 Microscopy: clue cells (bv); mobile trichomonads (trichamoniasis)	Metronidazole 2 000 mg PO stat • Follow up if not resolved in 1 week • STI testing for patient and partner
Foreign body	Discharge and itching; foreign body on exam	Remove foreign body; antibiotics not indicated

Disposition

Discharge if well and stable. Counsel on condom use. Refer for HIV and other sexually transmitted infection testing. Admit patients with haemodynamic instability, signs of sexual abuse or need for IV antibiotics.

192 Acute pelvic pain

Acute pelvic pain may represent benign or life threatening causes. Meticulous history and physical exam is key.

The first five minutes

ABC, VS, IV access – as needed.

History and physical examination

Key historical features

Ask about:

- Characteristics of pain: time course, progression, location, radiation, intensity, medications or other treatments used at home for pain, exacerbating or relieving factors such as intercourse
- The last menstrual period and type of contraception, vaginal bleeding, recent or current pregnancy, miscarriage, or abortion; associated features (vomiting, diarrhoea (frequency, presence of blood or mucus), dysuria, urinary frequency or urgency, fever)

Signs and symptoms

- Undertake a detailed examination of the abdomen and pelvis: Assess for suppleness, masses, tenderness and location of tenderness, discoloration or ecchymosis, prior surgical scars, guarding, rebound
- Speculum exam: vaginal lacerations or discharge (if present, note colour and odour), evaluate cervix for colour, friability, discharge; evaluate if open or closed
- Bimanual exam: CMT, uterine and adnexal tenderness, discharge

Possible causes and differential diagnosis

- Gastrointestinal: gastroenteritis, diverticulitis, appendicitis (right lower quadrant pain, may have associated fever, nausea, anorexia, or no associated symptoms), small bowel obstruction (abdominal pain and vomiting or nausea), inflammatory bowel disease
- OB: early pregnancy, ectopic pregnancy, miscarriage
- Gynaecologic: PID (discharge, CMT, uterine or adenexal tenderness), torsion (sudden onset severe pain, adenexal tenderness and/or mass on exam, testicular tenderness in male), endometriosis (recurrent, cyclical pain), ovarian cyst, fibroids (irregular, enlarged uterus), tubo-ovarian abscess (fever, pelvic inflammatory disease, positive US), buboes
- Urological: nephrolithiasis (suspect if blood in urine, colicky pain), urinary tract infection (dysuria or frequency + positive urinalysis)
- Hernia: inguinal, scrotal, femoral, umbilical (diagnose by examination)
- Other: sickle cell crisis, neurogenic, mesenteric thrombus, prostatitis, parasites, DKA

Investigations

- Labs: CBC, renal, urinalysis, vaginal smear ◇
- Imaging: US (ectopic pregnancy, ovarian cyst, free fluid (if present, consider ectopic or ruptured cyst), tuboovarian abscess, fibroid, torsion) ◇; CT abdomen ◈

Treatment

The goal of acute management is identification and management of serious conditions, analgesia and early directed therapy.

- If dehydrated or in shock, place IV, give 1–2 l IVF (crystalloid)
- Analgesia; if guarding or rebound on examination, consult surgery

Disease specific

- UTI: ciprofloxacin 500 mg BID ×3 days in women, 7 days in men; 14 days if pyelonephritis (alternative – co-trimoxazole)
- PID: (doxycycline 100 mg PO BID × 7 days AND ciprofloxacin PO 500 mg × 1) OR (ceftriaxone 250 mg IM × 1 AND metronidazole 500 mg PO TID × 7 days); if candidiasis, add fluconazole 150 mg PO × 1; if pregnant: erythromycin 500 mg QID × 7 days AND ceftriaxone 250 mg IM × 1; metronidazole if after 1st trimester; add nystatin
- Hernia: 📖 p. 253
- Ectopic pregnancy, threatened miscarriage (📖 First trimester vaginal bleeding, p. 480)
- Torsion: refer urgently for surgery
- Fibroids: 📖 Abnormal uterine bleeding, p. 478
- Endometriosis: oral contraceptives and NSAIDs, follow up with gynaecology
- Parasites: mebendazole (or other locally available treatment)
- Ovarian cyst: analgesia; if persistent pain, consult gynaecology or transfer
- Appendicitis, diverticulitis: 📖 Appendicitis, p. 234, Diverticulitis, p. 248

Disposition

Specific to underlying condition. Admit if peritoneal signs, unable to take oral fluids, severe pain, or concern for surgical aetiology.

193 Emergency treatment of sexual violence

Sexual violence occurs in all societies and can take place in any location, perpetrated by (and happen to) anyone, regardless of social status. Timelines to start ARV, STI, and pregnancy prophylaxis are critical. Management is multidisciplinary and should be integrated with the social and judicial system. 📖 Domestic and intimate violence victims, p. 907.

Definition

Sexual violence can be verbal, physical or emotional and occurs without the victim's consent. It includes rape, attempted rape, sexual harassment, and touching.

The first five minutes

- ABC, VS, IVF – as needed
- Check for and treat life threatening injuries

History and physical examination

Conduct in a safe, private environment; explain everything, reassure the patient.

Key historical features

Record the history as much as possible in the patient's own words. Determine date and time of the assault, number of assailants, type of assault. Ask about other physical injuries, date of last menstrual cycle, if on contraception.

Signs and symptoms

Examination should be performed in a calm and considerate manner; be careful not to force or pressure the patient. Always have a chaperone present (regardless of your gender). Know before starting what elements are crucial for the judicial system in your local context. Be guided by the local sexual assault kit.

- Head to toe examination: identify and describe any injuries (location, size, type, shape); note position patient was examined in
- Genital examination (including anal margin): describe injuries in clockwise direction; location, old or new
- Evaluate other sites, including lips and breasts
- Photograph injuries per local evidence laws with patient permission

Evidence collection

- Collect in accordance to local laws and capabilities
- Change gloves at each step; remember the patient can refuse any step
- Place all samples in labelled evidence envelopes, do not add other substances
- Collect all biological material for DNA testing if available: saliva, hair, semen, skin, blood, substances under nails, secretions; conserve patient's clothes

Investigations

Undertake additional focused investigations as needed.

- Labs: HIV, pregnancy test ◇; hepatitis B, LFT, (if source available – test for HIV, STD testing, hepatitis B, Viral load and CD4) ⟐

Management

The goal of acute management is to identify injuries, and mitigate potential consequences. Treat traumatic injuries. Complete or local legal documentation for all patients; if patient does not want it at that time, place in chart in case they eventually need it.

Disease prophylaxis

If anal or vaginal penetration or body fluid exposure.

Table 193.1 Disease prophylaxis

	Non-pregnant adults	Pregnant	Children
Syphillis	Penicillin benzathine 2.4 million IU IM × 1	Penicillin benzathine 2.4 million IU IM × 1	Penicillin benzathine 50 000 IU/kg IM ×1
Gonorrhea	Ceftriaxone 250 mg IM ×1 OR ciprofloxacin 500 mg PO × 1	Ceftriaxone 250 mg IM × 1	Cefixime 8 mg/kg PO × 1
Chlamydia	Doxycycline 100 mg PO BID × 7 days	Erythromycin 500 mg PO Q6h × 7 days	Erythromycin 12.5 mg/kg Q6h × 7 days
Trichomonas and BV	Metronidazole 2 g PO × 1	Metronidazole 2 g PO × 1 (after 1st trimester)	Metronidazole 5 mg/ kg PO TID × 7 days
Pregnancy: if ≤ 5 days after assault	Levonorgestrel 1.5 mg PO × 1; or 0.75 PO QD × 2 or IUD insertion within 5 days	None	If post-menarche: adult dosing Pre-menarche: none
HIV: if ≤ 72 hr after assault	28 day course. Per national guidelines or www.cde.gov/recommendations		
Tetanus	Last tetanus vaccination ≤ 10 years: none Last vaccination > 10 years + open wound: booster dose of tetanus IM		

	No vaccination + open wound: antitoxin + 1st vaccine in the other arm
Hepatitis B	If patient not already vaccinated: 500 UI immunoglobulin + vaccine.

Follow up testing: HIV at 6 weeks, 3 and 6 months; HbV at 3 months.

Psychological treatment

Ideally by psychologist/ trained social worker, but can also be done by doctor or nurse.

Legal

Encourage and support patient with reporting the assault to authorities. Social assistance may be needed. Referral to institutions or organisations that support victims of violence is essential.

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Part

4

L. Ophthalmology

194 The emergency eye examination

Eye complaints are common acute care problems. Even relatively minor injuries or illnesses, if managed poorly, can lead to visual loss with devastating implications. All patients with eye complaints require a complete ophthalmologic examination. Use a systematic anatomic approach, from the exterior to the interior of the eye.

Equipment

Essential items for a complete examination:
- Visual acuity evaluation chart (see 📖 Visual acuity, p. 958)
- Torch or other focal light source
- Patient's usual corrective lenses (or paper with a pinhole)
- Ophthalmoscope with cobalt blue capability ◇
- Eye drops: fluorescein, topical anaesthetic, mydriatic ◇
- Tonometer ◈
- pH testing paper ◇

The examination room should ideally have a dimmable light source and the means to be made completely dark. A slit lamp ◈ is a very valuable addition if available.

History

A complete exam begins with a thorough history, including:
- Timing: acute, chronic, insidious
- Quality: painful, painless, pruritic, photosensitive
- Visual acuity: complete loss, partial loss, colour-specific loss, blurriness
- Location of visual defect: centre, peripheral, specific quadrants of visual fields
- Discharge: watery, purulent
- Eyes involved: monocular or binocular
- Trauma: atraumatic, penetrating, blunt, foreign object
- Past medical history: previous eye problems, recent illness, HTN, DM

External eye examination

Examine the periorbital region (rashes, trauma, lymphadenopathy, oedema, erythema, and emphysema). Inspect and palpate eyelids, palpebral fissure and orbital rim (do not put pressure on the eye if you suspect globe injury). Inspect

conjunctiva and sclera; retract eyelids to facilitate inspection of the interior surfaces of eyelids. Inspect cornea (including fluorescein staining at the end of the exam) and the anterior chamber and iris.

The five vital signs of the eye

The emergency eye examination includes five essential components.

Pupils

- Size (average diameter 3–5 mm; difference between pupils < 1 mm)
- Shape (circular)
- Reactivity (normal versus sluggish versus absent) to direct and indirect light. Perform the 'swinging-flashlight test': both pupils should constrict to light in either eye. If both constrict to light in the L eye, but not to light in the R eye, there is a R 'afferent pupillary defect'
- Accommodation (have patient look at an object, then have them look at a much closer object in the same line of sight, observing for the convergence of pupils)
- If a pupil is unresponsive to light and accommodation, it is 'fixed'

Extraocular muscles and CN 3, 4 and 6

Standing approximately 1 m from the patient, and ask them to follow a target without moving their head. Slowly track movements in an 'H' pattern to evaluate all eight visual fields.

- Evaluate all extraocular movements
- Evaluate ptosis
- Evaluate eye position
- Evaluate nystagmus

Visual fields

Assess via the confrontation method: patient covers one eye and looks directly at the provider's nose (distance about 1 m). Provider slowly brings a moving finger into each of the four visual field quadrants; patient indicates at which point they can see the finger. Repeat for each eye.

Visual acuity

Quantitative measurement of the ability to focus on an object and detect details. Normal is (6/6 m) – how far away the patient can see the object/how far away an individual with normal acuity can see the object. With one eye covered, the patient reads each line – record the lowest line read. Testing should be done with patient's normal glasses on. An E chart (patient indicates direction) is good for illiterate patients and those not familiar with the Roman alphabet.

Intraocular pressure measurement

Contraindicated with penetrating eye trauma and corneal defects. Apply topical ocular local anaesthetic before performing the measurement. May be measured by tonometry or digital palpation over closed lid comparing to unaffected eye. Raised pressure implies glaucoma. Normal IOP is 10-20 mmHg. 40 mm requires lateral canthotomy. (See 📖 Glaucoma p. 514 and 📖 Lateral canthotomy p. 834.)

Additional examination modalities/tests

Ultrasound

Most useful for: ocular trauma, posterior chamber (retinal detachment, vitreous haemorrhage), dislocated lens, foreign body. Use the high-frequency (7.5–10 MHz) linear transducer, with water-soluble gel over the shut eye. Do not use if suspected globe rupture.

Slit lamp

Allows for a magnified exam of the lid, sclera, cornea, and anterior chamber. Useful for examination of the red eye. Patients should be awake, alert, and able to follow commands. Supplies needed include fluorescein strip, saline drops, and cotton swabs.

Fluorescein stain

Introduce fluorescein onto the eye (drops or strips wet with sterile saline). Examine the eye closely using a cobalt blue light source from an ophthalmoscope or slit lamp. The fluorescein will adhere to any corneal defect and appear bright green/blue with cobalt light. See image section.

Fundoscopic examination

An ophthalmoscope is a light source with an adjustable magnifying glass which improves examination of external and anterior chamber structures. The primary use is to examine the posterior chamber, particularly the retina. Dilation of the pupil is essential to complete examination.

Fundal exam by ophthalmoscope can diagnose papilloedema, retinal haemorrhage, chorioretinal lesions, retinal venous occlusion, diabetic retinopathy, and retinoblastoma.

195 Approach to the red eye

Red eye is a common complaint. There are many causes, ranging from relatively innocent (allergic conjunctivitis) to very dangerous (angle closure glaucoma). Thorough clinical evaluation will usually reveal the cause.

General diagnostic approach

Complete a thorough evaluation of the eye (see 🕮 The emergency eye examination, p. 508). Of particular importance is evaluation of visual acuity, looking for corneal damage and considering infection as a cause. The clinical pattern and associated features will usually suggest the cause.

Vision affected?	No: conjunctival process, corneal abrasion or foreign body, cavernous sinus thrombosis Yes: keratitis, iritis, or angle-closure glaucoma
Foreign body sensation?	Unable to keep eye open, corneal process Milder ‘scratchy feeling’ allergy, viral conjunctivitis, or dry eyes
Photophobia?	Suggests active corneal process, iritis, keratitis
Discharge?	Watery discharge: self-limiting process (allergy, hordeolum, viral conjunctivitis, allergic conjunctivitis, dry eyes) Opaque discharge: bacterial conjunctivitis, bacterial keratitis
Pupil reactive to light?	Fixed pupil in mid-dilation: angle closure glaucoma
Pattern of redness	Diffuse: suggests conjunctivitis Cilliary flush: infectious keratitis, iritis, or angle-closure glaucoma
White spot, opacity or foreign body on cornea?	White spot: bacterial keratitis Raised, grayish branching (dendritic) opacity: herpes simplex keratitis
Hypopyon or hyphaema?	Hypopyon: sight threatening infectious keratitis or endophthalmitis Hyphaema: penetrating eye injury, retinal detachment, and angle-closure glaucoma

Conjunctivitis

- *Causes:* conjuctival inflammation may be due to infection (usually viral; bacterial infection rare but dangerous, especially neonatal bacterial conjunctivitis), allergy, chemical irritation and auto-immune inflammation
- *Clinical:* cardinal symptoms of conjunctivitis are redness (viral and allergic usually bilateral), tearing, discharge (watery → allergy, mucoid → viral, bacterial: purulent), a foreign body sensation and superficial eye pain or itching. Visual activity usually unaffected
- *Treatment:* symptomatic treatment includes analgesia, lubricating eye drops and cold or warm compresses. Specific treatment depends on the cause. Allergic: allergen avoidance, antihistamines. Viral: symptomatic treatment and prevention of spread. Bacterial: topical antibiotics such as chloromycetin eye drops or ointment. In

children, high rate of bacterial suprainfection of viral conjunctivitis. Treat both with topical antibiotics

Subconjunctival haemorrhage

- *Causes:* traumatic or spontaneous (consider coagulopathy)
- *Clinical:* localised red/purple spot or area. May cause mild eye irritation or pain
- *Treatment:* ensure no penetrating eye injury or coagulopathy. Management is symptomatic. The discoloration fades over weeks to months

Corneal injury

- *Causes:* scratching of corneal epithelium – extended-wear contact lenses, sanding, grinding, sawing, other trauma
- *Clinical:* pain, tearing, photophobia, foreign body sensation, blurry vision. Fluorescein staining reveals the epithelial defect. Carefully search for a foreign body, especially on the internal surfaces of the eyelids and consider an infective corneal ulcer
- *Treatment:* analgesia, lubricating eye drops, topical antibiotics. Consider topical NSAID drops. Avoid home use of topical anaesthetic as increases risk of further injury

Iritis/anterior uveitis

- *Causes:* inflammation of the anterior uveal tract. Usually autoimmune but may also be viral, infiltrative process or idiopathic. HIV infection is strongly associated with anterior uveitis
- *Clinical:* ocular pain (particularly with induced miosis – light in eye or accommodation test), photophobia, ciliary flush, miosis, abnormal pupil shape or reaction. Look for cellular precipitates on the interior surface of the cornea and a hypopion (see image section)
- *Treatment:* analgesia, long acting mydriatics and urgent referral for ophthalmology evaluation. Consider HIV testing

Blepharitis

Inflammation of the eyelids due to infection or obstruction of eyelid glands.

Staphylococcal blepharitis/hordeolum (stye)

- *Causes:* arises from the glands to the eyelashes
- *Clinical:* discharge from eyelids, induration, crusting (see image section)
- *Treatment:* hot, moist eye packs, baby shampoo scrubs

Seborrhoeic blepharitis

- *Causes:* arises from Meibomian glands; associated with skin disorders (rosacea, eczema, dermatitis)
- *Clinical:* scaling of the eyelids
- *Treatment:* hot, moist eye packs and baby shampoo scrubs

Scleritis and episcleritis

- *Causes:* idiopathic, viral, association with autoimmune diseases
- *Clinical:* both present with eye pain and redness. Scleritis is more painful, episcleritis has a more acute onset. The redness of scleritis is usually fixed (episcleritis and conjunctivitis causes blanching redness)
- *Treatment:* analgesia with NSAIDs. Refer to ophthalmologist if severe, refractory or if the globe architecture seems abnormal

Acute angle closure glaucoma

📖 p. 516.

Cavernous sinus thrombosis

📖 p. 388.

Bacterial keratitis

- *Causes:* bacterial infection of the cornea. Rare. More common in contact lens wearers
- *Clinical:* eye pain and photophobia, redness, purulent discharge and FB sensation. The classic finding is a corneal opacity or infiltrate (typically a white spot, which stains with fluorescein) (see image section)
- *Treatment:* topical bactericidal antibiotics (ideally after obtaining cultures). Emergency referral to an ophthalmologist

Viral keratitis

- *Causes:* herpes simplex virus, varicella zoster virus, adenovirus. HSV keratitis is associated with a significant risk of corneal blindness
- *Clinical:* red eye, photophobia, foreign body sensation, and watery discharge. Diagnostic finding for HSV keratitis is a grey branching (dendritic) opacity, for adenoviral keratitis is multiple punctuate lesions; both stain with fluorescein
- *Treatment:* topical (trifluridine) or oral (acyclovir) antiviral agents. Urgent referral to an ophthalmologist

Hypopion

- *Causes:* any cause of ocular inflammation that involves the anterior chamber. WBC cells layered out in the anterior chamber appears as a pale sediment (see image section). Can be associated with uveitis, endophthalmitis, keratitis etc
- *Clinical:* red eye, altered vision
- *Treatment:* emergency referral to an ophthalmologist

Hyphaema

📖 p. 766.

196 Glaucoma

Glaucoma is ocular pathology (optic neuropathy with progressive loss of vision) caused by elevated intraocular pressure (IOP). Elevated IOP results from an imbalance in the production and drainage of aqueous humour. Glaucoma may be primary or secondary to trauma, infection, drugs and more. Consider glaucoma in all cases of eye complaints associated with GI complaints.

The first five minutes

Assess visual acuity and IOP. If evidence of acute angle closure glaucoma, rapidly initiate management below.

Angle closure glaucoma

The anterior chamber recess is narrowed and mechanical obstruction of aqueous humour results – increase in IOP is usually acute and severe. Often precipitated by mydriasis and accommodation (i.e. reading in poor light). Risk factors include older age and farsightedness.

History and physical examination

Cardinal signs are eye pain (usually unilateral) with abnormal vision and redness (often with cilliary flush). Additional features include: poorly reactive, mid-dilated pupil, nausea/emesis, corneal oedema, headache, abdominal pain.

Clinical diagnosis supported by tonometer measurement of elevated IOP (normal 10–20 mmHg); if no tonometer, digital palpation of globe through closed lid (may feel harder than unaffected eye). Do not palpate if any risk of globe rupture.

Management

The goal of acute management is to restore flow of aqueous humour (reverse angle closure), lower IOP, and reduce inflammation.

- Step 1: block aqueous humour production: topical beta blocker (timolol 0,5% one drop), alpha2-agonist (apraclonidien 1% one drop), acetazolamide (500 mg PO or IV)
- Step 2: facilitate outflow of aqueous humour: miotics (pilocarpine 1%–2%, one drop q 15 min × 2, then Q6h) – only effective once IOP < 40, usually about one hour after step one initiated
- Step 3: consider mannitol to reduce aqueous humor volume: 1 g/kg IV
- Step 4: reduce inflammation (1–2 doses topical steroids)

Urgent ophthalmology consult mandatory. It is essential to treat the associated symptoms effectively: analgesia and anti-emetics.

Open angle glaucoma

Most common form of glaucoma in Africa. Slow, chronic increased resistance to outflow to the canal of Schlemm via the trabecular meshwork with chronic progression of bilateral optic neuropathy. Risk factors: increased age, family history, elevated IOP. In addition to increased resistance to aqueous humour outflow, other potential secondary causes of open-angle glaucoma include inflammation, trauma, malignancy or drug-induced.

History and physical examination

Typically painless and asymptomatic until in advanced stages; progressive peripheral to central visual field loss.

Loss of peripheral visual fields; changes in optic nerve on fundoscopy; IOP may be in normal range.

Management

These patients require no emergency management. Prompt referral to an ophthalmologist should be made.

Differential diagnosis

- Angle closure glaucoma: any cause of a painful red eye (📖 p. 511) (uveitis, keratitis, conjunctivitis etc.)
- Open angle glaucoma: any cause of progressive visual loss (diabetic or hypertensive retinopathy, macular degeneration etc.)

Disposition

Admit all patients with acute angle closure glaucoma to the ophthalmology team. Patients with open angle glaucoma should be discussed with an ophthalmology service.

197 Periorbital and orbital cellulitis

Both periorbital and orbital cellulitis are typically unilateral and more common in children. Common causative organisms: *Staphylococcus aureus*, *Streptococcus pyogenes*, and *Haemophilus influenzae*.

Periorbital cellulitis

Superficial soft tissue infection anterior to the orbital septum, seldom leads to serious complications. Causes include primary infection or spread from adjacent structures (bacterial sinus infections, facial trauma, insect bites, and dental infections).

Differential diagnosis: herpes simplex or varicella keratitis, endophthalmitis, hordeolum, chalazion, allergic reaction, conjunctivitis, uveitis.

History and physical examination

Pain, redness, swelling and heat around the eye (usually unilateral); normal visual acuity, normal (painless) extraocular movement.

Investigation

Lab investigations and imaging unhelpful (except for complications).

Management and disposition

- Mild: Outpatient treatment:
  - » Amoxicillin/clavulanate 875/125 mg BID 7–10 days OR
  - » Cephalexin 500 mg Q6h 5 days OR
  - » Clindamycin 450 mg TID 5 days (penicillin allergy)
- Severe (or young children): admit, IV antibiotics

Orbital cellulitis

Deep tissue infection. Affects the content of the orbit (adipose tissue, extraocular muscles, nerves, blood vessels) and ultimately the eye. May lead to loss of the eye, extension to surrounding structures and sepsis.

A deep source (i.e. cellulitis, dental abscess) more common. Increased orbital pressure and direct infection leads to tissue ischaemia and destruction. Up to 10% result in loss of vision. May also spread to adjacent structures and cause meningitis, intracranial abscesses, cavernous sinus thrombosis, etc.

Differential diagnoses: as for periorbital cellulitis. Consider also cavernous sinus thrombosis (📖 p. 388), tumours, and TB.

History and physical examination

- Blurry vision; pain on eye movement; headache
- Eyelid erythema, warmth, tenderness, oedema; chemosis (oedema of the conjunctiva); decreased visual acuity; proptosis (bulging of the eye); limitation of extraocular movements, or pain with extraocular movement

Investigation

- Labs: blood culture ◇ prior to antibiotic treatment may help narrow ongoing antibiotic use
- Imaging: US ◇ may demonstrate an abscess;. CT orbits ◈ will visualise extent of disease: emergency if worsening visual acuity or blurry vision, severe proptosis, CNS involvement (severe headache, protracted vomiting, cranial nerve deficits), very young patients

Management and disposition

Prompt IV antibiotics:

- Vancomycin 15–20 mg/kg bid AND one of the following:
  - » Ceftriaxone 100 mg/kg QD, max 2 gm OR
  - » Ampicillin-sulbactam 200 mg/kg (max 3 g) d div Q6h OR
  - » Clindamycin 30 mg/kg/d (max 1 800 mg) div TID OR
  - » Ciprofloxacin 400 mg bid if penicillin allergic adult

Urgent ophthalmology consultation and consideration for surgery.

198 Approach to acute visual loss

Prompt and correct treatment may avert permanent sequlae in the patient presenting with acute visual loss. The algorithm below (Figure 198.1) is designed specifically with the African emergency care provider in mind.

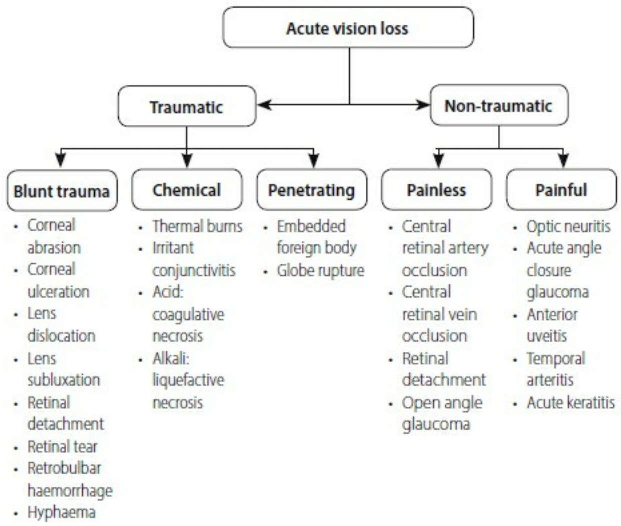

Figure 198.1 Acute visual loss algorithm

See 🕮 p. 764. for traumatic causes of acute visual loss.

Table 198.1 Non-traumatic causes of painless visual loss

Diagnosis	Cause/Risks	Findings	Diagnostics	Management
Central retinal artery occlusion	Often embolic (i.e. a stroke of the eye); vasculopathies	Unilateral painless vision loss. Often rapid and severe. Prominent afferent pupillary defect	Oedematous, pale grey retina, cherry-red fovea	Dislodge the embolus (digital globe massage over a closed eyelid for 10–15 seconds); hypercarbia (breathing into a paper bag for 10 minutes dilates retinal artery), lower IOP. Emergency ophthalmology consultation
Central retinal vein occlusion	Thrombosis or compression by nearby structures; vasculopathies, glaucoma, hypercoagulable states, vasculitis	Unilateral mild to severe loss of vision; often an afferent pupillary defect	Retinal haemorrhages, disc oedema	Emergency ophthalmology consultation. Lower IOP.
Retinal detachment	May be traumatic or atraumatic. Often associated with diabetes or inflammatory eye condition	Flashes of light, floaters, visual loss which is filmy, cloud-like or curtain-like	Retina appears out of focus Reduced visual fields	Emergency ophthalmology consultation
Open angle glaucoma	🕮 p. 516			
Transient ischaemic attack or stroke	CNS ischaemia, particularly of the occipital lobes	Cortical blindness: preservation of pupillary reflexes and a structurally normal eye	Stroke work up (🕮 p. 464)	Stroke management

Table 198.2 Non-traumatic causes of painful visual loss

Diagnosis	Cause	Findings	Diagnostics	Management
Acute angle closure glaucoma	p. 516			
Uveitis keratitis, endophthalmitis	pp. 511–513			
Optic neuritis (may also be painless)	Acute monocular vision loss caused by focal demyelination of the optic nerve	15–45 years. Vision loss over hours to days. Pain with eye movement	H&P most important. MRI brain with contrast	High dose methylprednisone *may* decrease development of multiple sclerosis over 2 years.
Giant cell (temporal) arteritis	p. 646			

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Part

4

M. Orthopaedics

199 Approach to fractures in adults and children

Fractures may be isolated or may be associated with injury of nearby vessels or nerves. Always consider whether the cause of the fracture may be a condition that requires treatment, such as seizure leading to fall.

The first five minutes

- ABCs – long bone or pelvic fractures may cause hypovolaemic shock; control haemorrhage with external pressure
- Assess neurovascular status
- Immobilising the fracture is important to pain control and restoring perfusion
- Analgesia

History and physical examination

Key historical features

- Mechanism (helps identify likely injury)
- Right or left hand dominance, if an upper extremity injury
- Stress fractures: consider in repetitive movement, athletes
- Consider non-accidental injury in children and vulnerable adults (see below)

- Pathologic fracture: bone tumours, bone cysts, rickets, malnutrition, kidney disease or osteogenesis imperfecta may weaken bone

Physical examination

- Look: bruising, swelling, deformity, abnormal movement or open wounds, joint dislocations or other fractures
- Feel: tenderness or crepitus, neurovascular status
- Move: range of motion – active (where possible) and passive

Describing fractures

- Right or left side
- Open *(compound)* fractures: bone exposed to environment. Identify by wound overlying fracture or bony fragments protruding through skin
  - *Type I* – wound < 1 cm, minimal tissue damage
  - *Type II* – wound > 1 cm, moderate tissue damage, no dead tissue
  - *Type III* – wound > 1 cm, extensive tissue damage, dirty wounds, GSW
- Simple (2 fragments) or comminuted (multiple bone fragments)
- Type:
  - Compression: fractures resulting from direct load, often comminuted
  - Wedge: compression fracture of the anterior aspect of vertebral body Burst: compression type fracture of the vertebral body with expulsed fragments, due to axial load
    Avulsion: fragment of bone is torn away from the main body of bone, typically at a tendon or ligament insertion
- Fracture lines may be transverse, oblique, diagonal, longitudinal, spiral or impacted
- Fractures may be angulated (the angle between the distal fragment and the axis of the intact bone), displaced (the position of the distal fragment relative to the proximal bone) or distracted (the distance between the fragment and its expected anatomic position)
- Extension to joint (intraarticular)

Special considerations in children

Children exhibit distinct fracture patterns due to softer, more compressible quality of growing bones and the presence of an open physis (growth plate).

Greenstick fractures are incomplete one-sided fractures of long bones due to a bending force. Torus (buckle) fractures are incomplete long bone fractures with circumferential buckling of the cortex due to axial loading force.

Growing long bones have a diaphysis (shaft), metaphysis (where bone flares), physis (cartilaginous growth plate), and epiphysis (secondary ossification centre). Growth occurs at the physis, which is prone to separation and fracture. Anatomic alignment is critical to physeal fracture healing. Improperly treated physeal fractures may result in limited bone growth.

Salter-Harris classification

Grades physeal fractures from IV, with risk of growth plate problems increasing with grade. The mnemonic SALTR may be used.

Straight across – type I
- Fracture line through physis, resulting in separation of epiphysis and metaphysis

Above – type II
- Fracture line through physis and into metaphysis

be**L**ow – type III
- Fracture line through physis, through epiphysis, intraarticular

Through – type IV

• Fracture line extends from articular surface through to metaphysis

Right through growth plate – type V, crush injury to physis

• Force through epiphysis across physis crushes growth plate.

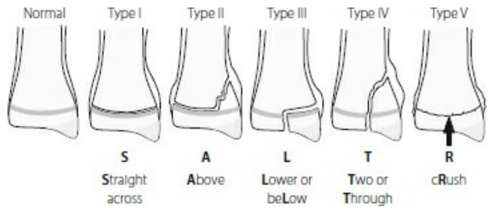

Figure 199.1 Salter-Harris classification of physeal features

Ossification centres

Fusion of ossification centres at different ages can make fractures difficult to recognise.

	Ossification centres	Approximate age of fusion
C	Capitellum	1 year
R	Radial head	3 years
I	Internal/medial epicondyle	5 years
T	Trochlea	7 years
O	Olecranon	9 years
E	External/lateral epicondyle	11 years

Non-accidental injury (NAI)

High suspicion for NAI in: any fracture in children <1 year; leg fracture in non-ambulatory child; bilateral long bone fractures; corner and bucket-handle fractures; posterior rib fractures; skull fractures; multiple fractures in different stages of healing; any fractures that do not correspond to patient history.

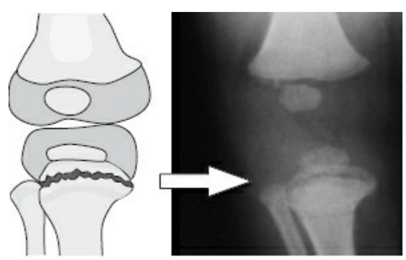

Figure 199.2 Bucket-handle fractures

Investigations

- X-ray ◇: at least two views – usually AP and lateral. Include XR of joints above and below. Low threshold for XR in children, as fractures are more easily missed, and ligament and tendon injuries are relatively uncommon
- Doppler ◇ or angiography ◈ for suspected vascular injury
- Bone scan, CT or MRI ◈ for high suspicion of X-ray occult fracture or soft tissue injury.

Management

- Assess and manage ABCs
- Splint initially to avoid compartment syndrome; place a cast once oedema resolved
- Early analgesia (consider regional nerve block, remembering to document neurological status prior)
- Tetanus toxoid 0.5 mg IMI for open fractures or open wounds

- For open fractures: broad spectrum antibiotic (usually first generation cephalosporins or vancomycin where MRSA is prevalent; for dirty contaminated wounds add gentamicin and metronidazole). Operative washout for Type II and III.

Critical documentation

Document serial neurovascular exams in all patients.

See also 🕮 p. 907 for special documentation considerations in non-accidental injury.

Disposition

- All open fractures require immediate orthopaedic consultation; Grade II or III open fractures require early operative washout.
- Displaced or severely angulated fractures require urgent orthopaedic consultation.
- Salter-Harris III, IV, and V fractures require urgent orthopaedic consultation
- Children in whom abuse is suspected should be admitted to the hospital until the child's safety at home may be evaluated.

200 Approach to FOOSH (fall on outstretched hand)

The first five minutes

Some injuries that result from FOOSH have a high risk of associated neurovascular injury. Early and serial exams of sensation and pulses distal to injuries are essential.

History and physical examination

Key historical features

While the characteristics of injuries associated with FOOSH vary with age, no age group is spared.

- ALWAYS ask about numbness, tingling, and subjective weakness.
- Young children almost never have carpal fractures but may sustain distal radial fractures, especially *torus* and *greenstick* fractures
- FOOSH in a toddler or young child may also result in a radial head or supracondylar humerus fracture (see 🕮 Special considerations for elbow injuries in children, p. 536)
- Adolescents and young adults are more likely to injure the carpal bones, especially the scaphoid. When they do sustain a distal radius fracture, it is often complex and associated with other injuries
- With increasing patient age, scaphoid fractures become less common, while distal radius fractures become more so

Signs and symptoms

- Carefully assess all bones and joints from the shoulder to the carpometacarpal joint, particularly the joint above and below the symptomatic area
- Look for bruising, crepitus, and limited ROM
- Do a focal motor and sensory exam
- Examine all pulses distal to the injury site.

Possible causes and differential diagnosis

Children

The differential diagnosis for FOOSH in children is:

- Buckle or torus fractures of the distal radius

• Greenstick fractures of the distal radius
• Distal radius fracture
• Supracondylar humerus fracture
• Radial head fracture
• Clavicle fracture or dislocation

FOOSH in adults

The differential diagnosis for FOOSH in adults is:
• Distal radius fracture, typically Colles
• Scaphoid and other carpal fractures
• Scapholunate and perilunate dislocation
• Distal radio-ulnar dislocation (Galleazi fracture)
• Monteggia fracture
• Supracondylar fracture
• Proximal humerus fracture (in elderly)
• Clavicle fracture

Investigations

X-ray ◇ symptomatic area as well as joint above and below. Dedicated views may be needed to evaluate the wrist and clavicle. Indirect findings (such as fat pad signs on wrist and elbow X-rays) may be helpful for diagnosis of upper extremity fractures.

Management

The goal of acute management is restoration of normal anatomical relationships to ensure perfusion and immobilisation of fracture fragments to reduce pain and prevent further damage to surrounding tissues.
• Provide analgesia
• Immobilise the affected area with a splint or sling
• Manage the associated injury
• Immediate orthopaedic consult ◇ is indicated for any neurovascular abnormality

201 Shoulder dislocation

History and physical examination

Key historical features

Mechanism may help predict type. Always ask about numbness, tingling, weakness, and any history of prior dislocation.
• Anterior dislocations by far the most common type; usually result from forced external rotation with abduction in the extended arm.
• Posterior dislocations rare injuries; result from anterior blow or violent contraction during seizures; often missed in patients with AMS.
• Inferior dislocations (luxatio erecta) occur when humeral head is forced below the glenoid fossa; very rare and have a distinct clinical presentation.

Signs and symptoms

Anterior dislocation

• Arm held in slight abduction and external rotation with limited ROM
• Prominent acromion process

• Shoulder has a 'squared-off' appearance and anterior shoulder appears full.

Posterior dislocation

• Prominent posterior shoulder and coracoid, with anterior flattening
• Limited abduction and no external rotation.

Inferior dislocation (Luxatio erecta)

• Arm locked overhead in 110–160° of abduction, elbow usually flexed with forearm resting on top of the head
• Humeral head may be palpable inferiorly along the lateral chest wall
• 60% associated with neurologic dysfunction.

Investigations

• XR: AP, lateral and Y views. The humeral head is normally centered on the midpoint of the Y (the Mercedes Benz sign)
• Anterior dislocation: infero-medial displacement of humeral head on AP. Other associated injuries include Hill Sachs deformity (compression fracture of the humeral head), Bankart lesion (avulsion fracture of the glenoid labrum) and avulsion fracture of the greater tuberosity
• Posterior: look for lightbulb sign (internally rotated humeral head appears spherical) or rim sign (overlap of glenoid fossa with humeral head)
• Inferior: humeral head beneath glenoid or coracoid.

Management

The goal of acute management is early restoration of the anatomic position of the humeral head, recognition of other injuries associated with dislocation or reduction, and immobilisation to prevent recurrent dislocation.

Analgesia can be by GA, regional block, procedural sedation and/or local anaesthesia infiltrated in the joint. Muscle relaxation may be crucial for reduction; benzodiazepines preferred for procedural sedation.

Options for reduction

• Scapular manipulation: quickest and safest method, repositions glenoid fossa for reduction. With patient upright, or prone with arm hanging off table, apply downward traction. Rotate inferior tip of scapula medially with direct pressure while rotating superior and medial edges lateral.
• Traction techniques: *Stimson*: patient prone, 3–5 kg weight at wrist. Apply gentle internal and external rotation with traction; reduction should occur within 20 minutes. *Milch*: with patient prone, place one hand in patient's axilla and other hand holding the patient's hand. Gently abduct arm fully, then externally rotate and apply gentle traction for reduction.
• Hippocratic: with arm in slight abduction, axial traction-countertraction is applied via sheet wrapped around the chest and under axilla.
• Kocher: elbow flexed to 90° adducant, slowly rotate forearm outward until resistance is felt; lift forward in the sagittal plane as far as possible. Reduction achieved by internal rotation, bringing hand to opposite shoulder.

Posterior dislocation

• Urgent orthopaedic consult ◇ when available
• Closed reduction may be attempted under procedural sedation. Reduction uses in-line traction-countertraction, gentle pressure on the humeral head, and slow external rotation of the arm

Inferior dislocation (luxatio erecta)

• Urgent orthopaedic consult when available ◇, as open reduction may be required and associated injury to the brachial plexus and axillary vessels common
• Closed reduction may be attempted under procedural sedation. Traction applied in line with the humeral shaft while an assistant applies countertraction. Gentle abduction usually reduces the dislocation

Immobilisation in a sling

- Traditionally done with arm in internal rotation. Immobilisation in external rotation reduces rate of recurrence but is difficult to achieve. Duration of immobilisation is dependent on age of patient and the complexity. Varies from 2–3 weeks in older patients, up to six weeks in younger patients with first dislocation

Other considerations

- Complex cases (associated fracture of humerus, rotator cuff tear, axillary nerve injury or recurrent dislocations) should receive early orthopedic follow-up and surgical intervention ◇
- Rehabilitation is key. Early shoulder exercise prevents adhesive capsulitis

Critical documentation

Document serial neurovascular exams, evaluate for humeral head fractures.

Disposition

In rare cases, admission required for operative management.

202 Shoulder and humerus injuries

See also 🕮 Shoulder dislocation, p. 530, and Elbow injury, p. 532.

History and physical examination

- Detailed history including age, hand dominance, co-morbidity, prior functioning
- Examine surrounding bony prominences, muscles, and soft tissue, giving particular attention to the joints above and below the symptomatic area
- Neurological examination – radial nerve injury is most common with midshaft humerus fractures and results in weakness of wrist, finger, and thumb extension
- There is a high risk of nerve injury in the elderly
- Vascular injury is less common, but bilateral pulses and capillary refill should be evaluated and documented

Possible causes and differential diagnosis

Fracture, muscle contusion or sprain, rotator cuff tear, brachial plexus injury, infection, disruption of the axillary artery, neuropathic arthropathy, and thrombosis of the axillary vein.

Proximal humerus fractures

- Tend to occur in the elderly (due to the effects of osteoporosis), or in young people as a result of severe trauma. Essential XR views: true AP (in the plane of the scapula), Y view and axillary view
- Management: immobilise in a sling. Non-displaced fractures involving one of the tuberosities may be definitively managed with a sling only. Early orthopaedic follow up ◇ as most will require open reduction and internal fixation ◇. Some osteoporotic fractures with multiple fragments require prosthetic shoulder replacement ◈

Fractures of the humeral mid-shaft

Various classifications exist but all fractures should be characterised by location, type, fragment position and orientation, and any associated injuries of nerve, vascular, or soft tissue structures.

- XR: AP and lateral; include the shoulder and elbow joints
- Immobilise in a traction splint to stabilise length: coaptation splints, valpeau dressings, hanging casts (for angulated fractures with minimal swelling), or functional bracing ◇. Hanging arm casts require sleeping upright and may overdistract fracture and be associated with non-union. Surgery ◇ indicated for failure of closed reduction, intra-articular extension, vascular compromise, segmental fractures, some pathologic fractures, open

fractures, bilateral humeral shaft fractures, and periprosthetic fractures

Fractures of the distal humerus

Classified by involvement of the articular surface and the lateral or medial columns. The commonest mechanisms include road traffic crashes, falls and direct trauma to the elbow.

Standard AP and lateral XR. CT scan may be helpful when the articular surface is involved ⟐. Most require open reduction and internal fixation ◇.

Clavicle fractures

Commonly result from a FOOSH in the elderly and high impact injury in the young.

Allman classification: middle (Type I), distal (Type II), and proximal (Type III). Mid-shaft fractures are the most common, followed by distal. Look for bruising and tenting of the skin and palpate for tenderness or step off. Assess for associated brachial plexus and artery injury.

Management is analgesia and sling ± swathe immobilisation for 1–2 weeks. Even while immobilised, early shoulder exercise is recommended, but arm should not be abducted above the shoulder in the early phases. The incidence of non-union is high in significantly displaced or shortened middle third fractures, and these should be referred to orthopaedics for potential surgical management ◇.

Acromioclavicular joint injury

Usually occur with falls onto the adducted shoulder. Patients present with tenderness over the AC joint, deformity (palpable step between distal clavicle and acromion) and prominent acromion depending on the severity. Pain is usually worse when crossing the arm to touch the opposite shoulder. On X-ray may have widening of the AC joint greater than 3 mm and widening of the coracoclavicular distance greater than 13 mm. Stress views are not recommended.

Management is by ice, analgesia (usually NSAIDS) and immobilisation in a sling until pain resolves. Prognosis is good. Orthopaedic follow up recommended for subluxation or dislocation of AC joint.

Rotator cuff injuries

Common cause of shoulder pain. May be caused by acute traumatic tears, inflammatory (adhesive capsulitis), or degenerative processes (impingement syndromes). Patients complain of pain over the deltoid area, worsened by abduction above shoulder level. Pain is often worse at night. On examination range of motion is preserved but there is pain with active abduction. X-ray only used to exclude bony injuries. Management is with ice, analgesia (NSAIDS) and immobilisation in a sling.

203 Elbow injuries in adults

Elbow injuries are common in upper extremity trauma and are not always obvious on initial X-ray imaging. Careful examination is essential.

The first five minutes

- Assess for open wounds and control bleeding
- Assess neurovascular status
- Provide analgesia
- Immobilising the fracture in a splint or sling is important to control pain and restore perfusion

History and physical examination

Key historical features

- Mechanism (helps identify probable injury)

• Right or left hand dominance, if an upper extremity injury
• Stress fractures: consider in repetitive movement, athletes
• Consider non-accidental injury in children and vulnerable adults
• Pathologic fracture: bone tumours, bone cysts, rickets, malnutrition, kidney disease or osteogenesis imperfecta may weaken bone

Signs and symptoms

• Look: bruising, swelling, deformity, abnormal movement or open wounds, joint dislocations or other fractures
• Feel: tenderness or crepitus, neurovascular status
• Move: range of motion – active (where possible) and passive

Investigations

• XR: AP and lateral.

The anterior humeral line should run through the middle third of the capitellum. Misalignment suggests a supracondylar fracture. While a posterior fat pad is always pathologic, a bulging anterior fat pad (sail sign) may also suggest an occult fracture.

Figure 203.1 Sail sign
Source: © Eric Silman, MD

Elbow fractures

Supracondylar fractures

Distal third of humerus fractures. Commonly the distal segment is displaced posteriorly. Typically occurs in patients > 50 years. Often caused by FOOSH. Associated median, radial and ulnar nerve palsies are common; document the patient's neurovascular status before and after any immobilisation. Undisplaced fractures may be treated conservatively with an above- elbow cast, displaced fractures should be referred for ORIF after immobilisation ◇.

Olecranon injuries

Typically occur after direct trauma to the elbow. Tenderness over the olecranon and inability to extend the elbow against pressure is highly suggestive of a fracture. Often complicated by ulnar nerve injuries. Undisplaced fractures may be managed in an above-elbow cast with 45–90° of elbow flexion. Early orthopaedic follow-up. Displaced fractures or those associated with elbow dislocation require urgent referral for ORIF ◇.

Radial head fractures

Radial head fractures are common. They may be caused by FOOSH with the elbow slightly flexed or direct trauma to the elbow. Often present with tenderness over the radial head and painful extension of the elbow. Nondisplaced fractures may be treated with analgesia and a sling for 5–7 days. Displaced, angulated or comminuted fractures require immobilisation and referral ◇.

Elbow dislocations

The elbow is second only to the shoulder as the major joint most frequently dislocated. Most dislocations are posterior. Most patients without associated fractures have an excellent long-term outcome.

History and physical examination

The classic mechanism of injury is a fall onto the outstretched hand with the elbow extended.

- Marked deformity, a prominent olecranon
- Elbow is 'locked' in about 45° of flexion
- Large joint haematoma
- The median nerve and brachial artery are at particular risk. Serial neurovascular examinations are crucial

Management

Treatment is prompt reduction. Patients may require procedural sedation or intra-articular anaesthesia.

- Reduce a posterior dislocation by stabilising the humerus and applying gentle traction to the wrist, then flex the elbow
- Assess and document neurovascular status post-reduction
- Move the elbow through flexion and extension to ensure stability
- Obtain post-reduction films
- Immobilise the elbow in 90° of flexion with wrist pronated
- Observe in hospital for stability of reduction and neurovascular checks

204 Special considerations for elbow injuries in children

Injuries to the elbow and distal humerus are common in children. These injuries may be subtle and result in long-term sequelae if not treated. See also 🕮 Elbow injuries in adults, p. 532.

The most common cause is FOOSH. The appearance of the ossification centres may make fractures difficult to

diagnose. (See also 🕮 Approach to fractures in adults and children, p. 522.)

The first five minutes

- Assess for other injuries
- Immobilise the elbow in a splint or sling
- Assess for open wounds and control bleeding; check neurovascular status
- Early analgesia

History and physical examination

Key historical features

- Mechanism of injury
- Position of arm and direction of force applied
- Ability to move arm after injury
- Pre-injury function, prior injuries or surgery

Signs and symptoms

- Swelling (effusion), tenderness, deformity or open wounds
- Test active range of motion (have the child reach for a toy held in different positions); if limited, test passive range of motion
- Test distal pulses, capillary refill, and sensation (specifically test radial, median, ulnar nerve motor and sensory function)

Possible causes and differential diagnosis

Supracondylar fractures

Supracondylar fractures are common and have a high risk of complications.

- XR: evaluate displacement, angulation and shortening. Assess the anterior humeral line (line along the anterior humeral margin). It should bisect the middle of the capitellum. The radiocapitellar line runs through the axis of the radius and should point at the capitellum

Figure 204.1 Supracondylar fracture

Management

- Serial neurovascular examination; analgesia
- Splinted in an above elbow splint with the elbow at 90° and forearm in neutral
- For undisplaced fractures with normal neurological examination, the child may be discharged and brought back

for early orthopaedic follow-up ◇
- For displaced fractures or evidence of neurovascular compromise, urgent referral to orthopaedics for surgical fixation ◇

Epicondylar fractures

Isolated fractures of either the lateral or medial epicondyles are usually due to FOOSH. Carefully assess neurovascular status. Immobilise in a posterior splint at 90°, provide analgesia and arrange orthopaedic follow-up ◇.

Radial head subluxation

Often called *pulled elbow or nursemaid's elbow*, it is common aged 1–3 years when a traction force is applied to the arm with the elbow extended and the wrist pronated, resulting in tearing of the annular ligament. The child refuses to move the elbow, which is held in a flexed and pronated position. Management – gentle rapid hyperpronation and flexion of the elbow. This does not require sedation, but it is important to explain the procedure to the parents first! Pre- or post-XR are not required if typical history and function.

205 Forearm and wrist fractures

Wrist injuries are common and may lead to significant long-term complications if untreated.

The first five minutes

- If open wound, stop bleeding
- Assess neurovascular status, including pulses, capillary refill and sensation
- Splint fracture; analgesia

Key historical features

Fractures are commonly caused by FOOSH or direct trauma.

Signs and symptoms

- Look for swelling, deformity and open wounds
- Evaluate the location and degree of pain, tenderness and limitation of movement
- Assess neurovascular status. Test median, radial and ulnar motor and sensory function (see 🕮 Hand examination, p. 542).

Investigations

Wrist and elbow XR: AP and lateral views with hand in neutral position.

Radial injuries

Distal radius fractures are the most common upper extremity fracture. There are a number of named fractures of the distal radius, but it is more important to identify and describe the injury in terms of location, displacement, angulation, and comminution.

Colles fracture

Most commonly caused by FOOSH. Typically characterised by a transverse fracture of the distal radius with dorsal displacement and angulation, impaction and an ulna styloid fracture.

Clinically patients may demonstrate the 'dinner fork' deformity of the wrist. Look for associated median nerve palsy.

Reduction of the Colles fracture may take place with procedural sedation or local anaesthetic.

- Disimpact the fracture by supplying axial traction – pull on thumb and ring finger with assistant holding the

elbow. Pull the distal segment back, up, and out to increase the deformity (allowing the periosteal ligaments to relax). With traction and the arm pronated, use both thumbs on the distal fragment and push down and outwards – moving the distal fragment volarly and towards the ulna

- Immobilise the arm in a well-moulded cast in full pronation, full ulnar deviation and slight flexion
- Post-reduction films should always be obtained. Aim to correct dorsal angulation and maintain radial length
- Orthopaedic follow-up ◇

Figure 205.1 Colles fracture

Source: © Eric Silman, MD

Smith’s fracture (volar angulation)

The Smith’s fracture (or reverse Colles’ fracture) is caused by a direct blow or fall on the dorsum of the wrist. Treatment is similar to Colles fracture.

Radial styloid fracture

The chauffeur fracture (or Hutchinson fracture) is a break in the radial styloid. Often due to direct impact on the radial side of the wrist. Management is immobilisation in a below elbow cast. If markedly displaced ORIF may be required.

Radioulnar joint injuries

Injuries to this joint are commonly missed in the acute setting. Dislocation of the ulna may occur with distal radius fractures. Occasionally ulnar dislocation at the radioulnar joint occurs without bony injury. Typically this is due to falls or sudden distraction or rotation.

History and physical examination

- Swelling of the wrist particularly on the volar aspect
- Ulna styloid prominence is absent
- The forearm is fixed in supination
- Assess neurovascular status.

Management

- Immobilise in below elbow cast
- Analgesia
- Refer orthopaedic surgery for reduction.

Forearm fractures

Isolated ulna fractures

These fractures typically occur due to direct trauma. Sometimes called nightstick or defensive fractures, they usually

occur when the arms are raised to protect the head. These are stable fractures unless they are significantly angulated or more than 75% displaced. Stable fractures should be immobilised in an above-elbow cast. Orthopaedic follow-up is required for unstable fractures.

Galleazzi fracture-dislocations

This rare fracture results from a FOOSH with the hand in pronation. There is a fracture of the middle to distal third of the radius with associated distal radio-ulnar disruption (dorsal displacement of the ulnar head). The arm should be immobilised in an above-elbow cast with the hand in supination. Urgent orthopaedic consult should be obtained for operative fixation.

Figure 205.2 Galleazi fracture

Source: © Eric Silman, MD

Monteggia fracture dislocations

This fracture usually results from a FOOSH with forced pronation or direct trauma to the dorsal ulna. There is a fracture of the proximal third of the ulna and disruption of the proximal radioulnar joint (radial head displacement). Distal neurovascular injury is more common than with Galleazi fractures. The arm should be immobilised in an above elbow cast with the hand in supination. Urgent orthopaedic consult should be obtained for operative fixation.

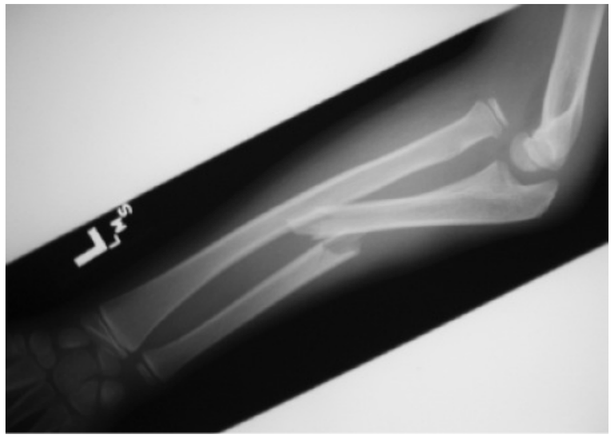

Figure 205.3 X-ray of Monteggia fracture
Source: © Michelle Lin, MD

206 Hand examination

Expose the hand and wrist to the elbow. Ask the patient for tender points.

Look

Inspect hands at rest, in the position of function (fingers flexed). In prone position: If finger extended consider flexor tendon injury. In supine position: if finger extended consider extensor tendon injury. If fingers malaligned consider fracture with rotation.

Look for swellings, deformities and muscle wasting. Ask patient to make a fist and extend fingers to get overall impression of function.

Feel

Check radial and ulnar pulses, capillary refill time. Feel the muscle bulk in the thenar and hypothenar eminences. Check for tendon thickening. Squeeze over the row of MCP joints, palpate the MCP joints and the intercarpophalyngeal joints and interphalyngeal joints. Feel web spaces. Assess the sensation over the areas supplied by the radial, ulnar and median nerves:

- Radial: dorsal web space of thumb and index finger
- Ulnar: volar tip of little finger
- Median: volar tip of index finger

Move

Assess:

- Radial nerve: wrist extension and finger extension
- Ulnar nerve: intrinsic muscles of the hand, spread fingers against resistance
  - Froment's sign – patient holds a piece of paper between their thumb and index finger (adductor pollicis). In ulnar nerve palsy the interphalangeal joint of the thumb will flex to compensate.
- Median nerve: thumb abduction while hand is flat on the table (abductor pollicis)

Assess all other movements and pay attention to specific muscle groups:

- FDS – individually tested by holding other fingers in hyperextension
- FDP – tested by fixing the PIPJ and thus isolating the DIPJ
- EPL – tested by asking patient to lift thumb up off a table while hand held palm down on table

Perform a functional assessment – power grip around examiners middle and index fingers; pincer grip against examiners index finger; picking up a small object.

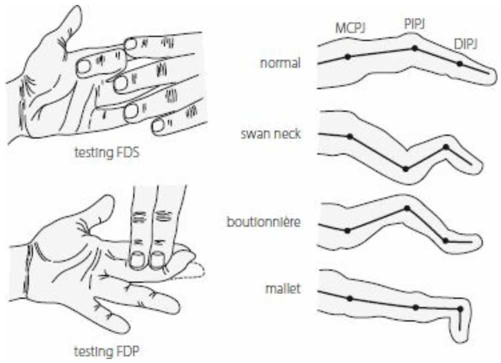

Figure 206.1 (a) Testing of superficial and deep tendons (b) Finger deformities

Source: Brown & Wyatt. 2008. *Oxford American Handbook of Emergency Medicine*. By permission of Oxford University Press, USA.

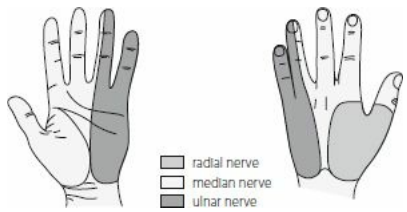

Figure 206.2 Innervation of the hand

Source: Brown & Wyatt. 2008. *Oxford American Handbook of Emergency Medicine*. By permission of Oxford University Press, USA.

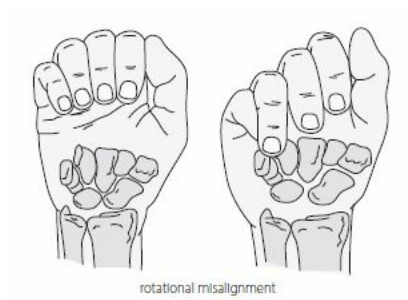

Figure 206.3 Rotational misalignment

Source: Brown & Wyatt. 2008. *Oxford American Handbook of Emergency Medicine*. By permission of Oxford University Press, USA.

207 Carpal fractures and dislocations

Carpal bone fractures account for about 20% of all injuries to the hand and wrist.

Scaphoid fracture

60–80% of all carpal fractures. The scaphoid forms a bridge between the carpal rows. Fractures may be very difficult to visualise on initial X-ray and clinical examination is essential.

Figure 207.1 Scaphoid fracture XR
Source: © Michelle Lin, MD

Clinical signs include tenderness over snuffbox, pain with axial compression of the thumb, or pain on palpation of the radial tubercle. In presence of clinical signs, treat as if fractured even with negative XR. Immobilise in thumb spica for 7–10 days and bring back for repeat XR and orthopaedic review ◇. Failure to immobilise leads to a high incidence of malunion and avascular necrosis.

If a fracture seen on XR immobilise in a thumb spica and arrange orthopaedic follow up ◇.

Triquetral fracture

Triquetral fractures, the second most common type of carpal fracture, occur with both direct trauma and with FOOSH injuries. Dorsal chip fractures are due to hyperextension when the ulnar styloid is jammed into the triquetrum. This produces tenderness just distal to the ulnar styloid. Fracture through the body is associated with perilunate, scaphoid and ligamentous injury. Patients with triquetral fractures require a below elbow splint and early referral to orthopaedics ◇.

Scapholunate injury

These are rare but usually caused by FOOSH. Patients present with tenderness over the scapholunate joint or instability of the scaphoid (scaphoid shift).

On the XR: a widened scapholunate gap (‘Terry Thomas’ sign), this gap can be increased by taking the XR with the fist clenched.

Figure 207.2 Scapholunate dislocation
Source: © Michelle Lin, MD

Lunate and perilunate dislocations

These are easily detected on the lateral wrist XR.

- Perilunate: the capitate will no longer sit in the same line with the lunate, midshaft radius, and metacarpals – it will sit outside the cup of the lunate.
- Lunate: further disruption leads to lunate dislocation. The lunate is noted to be 'spilling over' toward the palm, rather than cupping the capitate ('spilled tea-cup sign'). In the lateral and PA view the lunate is triangular rather than normal quadrangular appearance.

Management includes analgesia, immobilisation and urgent orthopaedic review ◇. Complications include avascular necrosis of the lunate (Keinbock's disease).

Figure 207.3 Lunate dislocation
Source: © Michelle Lin, MD

208 Fractures and dislocations of the hand and foot

Hand and foot injuries are common and may result in severe functional morbidity if untreated.

Anatomy

Each hand and foot has 19 bones: five metacarpals/metatarsals and 14 phalanges. Each finger and toe has three phalanges; the thumb and great toe each have two phalanges. Each joint in the hand is named and abbreviated to define the two bones involved:

- Metacarpophalangeal joint (MCP): between the metacarpal bone and the proximal phalangeal bone
- Metatarsophalangeal joint (MTP): between the metatarsal bone and the proximal phalangeal bone
- Proximal interphalangeal joint (PIP): between the proximal and middle phalagneal bones
- Distal interphalangeal joint (DIP): between the middle and distal phalangeal bone
- Interphalangeal joint (IP): between the two phalanges of the thumb or great toe.

History and physical examination

See 🕮 Hand examination, p. 542.

- Identify potential functional impact of injury: hand dominance, occupation, social support
- Look for pain, swelling, deformity, limited range of motion. Carefully examine each joint for instability or decreased range of motion, which may indicate a dislocation. Palpate each bone for points of maximal tenderness. Check neurovascular status distal to the injury. **Fractures and dislocations often have coexisting tendon and ligament injuries**

Investigations

- XR can help delineate anatomy of dislocations and fractures. Obtain at least two views: AP and lateral ◇

Management

- The goal of acute management is recognition of all injuries, stabilisation, analgesia, and appropriate referral where specialised management is needed to ensure a good functional outcome. Have a low threshold for orthopaedic consultation
- To facilitate thorough examination, pain control is paramount. See 🕮 Digital and Regional nerve blocks, p. 806 but detailed neurological examination should be performed prior to anaesthesia

Fractures

- Intra-articular fractures and fractures with significant displacement or rotation may result in deformity and impaired function. Orthopaedic referral ◇
- Reduction may be necessary to restore normal anatomical alignment and facilitate proper healing

Dislocations

- Disruption of ligaments connecting allowing disruption of normal joint anatomy
- Often have coexisting tendon injury
- Reduction should be attempted in order to restore normal anatomical position:
  - Re-create the injury: distract the two bones (pull them apart in the longitudinal plane)
  - Realignment may require flexion or extension at the joint after the bones are distracted
- Reduction can be verified by stability of the joint, restoration of normal range of motion, and radiographic alignment
- Verify neurovascular status after any reduction

Splinting

Anatomical position can be maintained by applying a splint. Proper splinting will facilitate healing and restoration of function to the injured hand or foot.

- Immobilise the bone above and below the dislocated joint or fractured bone
- Do not apply a splint too tightly. Injured extremities will swell over time. Instruct patients to loosen any dressings that feel too tight to prevent compartment syndrome
- Verify neurovascular status is intact after applying a splint
- Fingers and toes can be ‘buddy taped’ together using the uninjured digit as a splint
- Place padding between digits to protect the skin
- If a tendon injury is present, splint the joint with the ends of affected tendon in closest proximity and refer to an orthopaedic/hand surgeon ◇

Splinting hands

- Splint the hand with the DIP and PIP joints in extension and the MCP joints flexed to 90°
- Splint the wrist in slight extension, and not in flexion (this can result in neurovascular injury)
- Consider using a sling

Splinting feet

- If the entire foot requires splinting, splint with the ankle in 90° flexion
- For metatarsal fractures, use crutches (if available) and do not bear weight

209 Hip fractures and dislocations

Hip fractures are associated with high morbidity and mortality due to lack of mobility post-injury.

The first five minutes

In elderly patients, hip fractures may follow falls caused by serious medical events such as CVA or MI. Post-fall, patients may be immobile for long periods resulting in hypothermia, dehydration or sepsis. Always do an initial general evaluation.

- ABCs
- Check distal pulses and sensation
- Check for dislocation and immobilise the affected leg (re-check neurovascular status after immobilisation) – analgesia
- If altered, check glucose

History and physical examination

Key historical features

- Unable to weight-bear
- Mechanical fall or found down?
- Recall of events? Any chest pain, DIB, or any chance of syncope?

Signs and symptoms

- Leg held in a fixed position, neck of femur fractures may be shortened and externally rotated
- Limited range of motion
- Assess neurovascular status

Investigations

- Labs: CBC, electrolyte, renal, ECG, type and cross ◇

• Imaging: CXR: AP pelvis XR, lateral hip XR
• Assess Shenton's line (continuous line along the inside of the femoral neck and internal pubic ramus) and the trabecular pattern of the femoral neck, disruptions in these may indicate subtle fractures

Hip fractures

Fractures of the proximal femur are divided into intracapsular (femoral head and femoral neck) and extracapsular (intertrochanteric, trochanteric or subtrochanteric) fractures. All require urgent orthopaedic review.

Fractures of the femoral head are rare. They are usually due to a direct force and associated with pelvic fracture or hip dislocation.

Femoral neck fractures

High risk of avascular necrosis, require early fixation (ideally within six hours).
Garden classification:
• Type I: incomplete or impacted fracture, inferior cortex intact
• Type II: complete but undisplaced, fracture line through superior and inferior cortices
• Type III: partially displaced complete fracture
• Type IV: grossly displaced or rotated complete fracture.

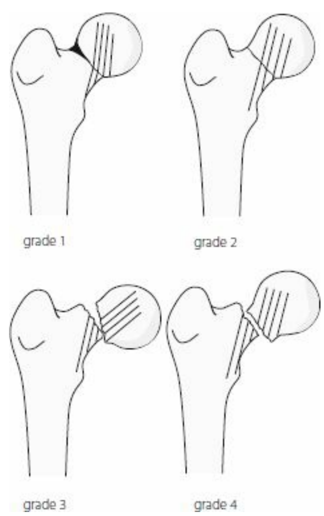

Figure 209.1 Garden classification of hip fractures

Intertrochanteric fractures

Fracture lines run between greater and lesser trochanters. May be stable (undisplaced) or unstable (displaced). Often difficult to diagnose on XR. CT or MRI may be needed ⟐. Complications are common.

Isolated trochanteric fractures

Avulsion fractures of either the greater or lesser trochanters may occur with forceful contraction of the gluteus medius (greater) or iliopsoas (lesser). Management is conservative but orthopaedic follow up advised. Significantly displaced greater trochanteric fractures in young patients may require surgical fixation ⟐.

Subtrochanteric fractures

Occur within 5 cm of lesser trochanter. Associated with minor trauma coupled with bony pathology such as metastatic disease or Paget's disease of bone in older patients. In younger patients, great force is required to break the bone. Present with significant pain and thigh swelling. Traction to the leg may relieve pain and minimise blood loss.

Management of hip fractures

- Immobilise with splinting or traction
- Analgesia
- Refer orthopaedics for internal fixation or hip replacement ◇
- Only undisplaced isolated trochanteric fractures may be treated conservatively (analgesia, non-weightbearing).

Hip dislocations

Hip dislocation is usually unilateral. The femoral head may lie anterior (10%) or posterior (90%) to acetabulum.

Physical exam

- The hip is commonly held in adduction and either internal (posterior dislocations) or external rotation (anterior dislocations)
- The femoral head may be palpable in groin (anterior dislocation)
- The leg is shortened with limited range of motion
- Assess and document the neurovascular status
- High-energy injury. Check for other injuries!

NB: Relatively minor forces may result in dislocation of artificial hips. Requires orthopaedic referral.

Management

True orthopaedic emergency. Early reduction leads to better outcome. May lead to avascular necrosis.

Reduce under procedural sedation.

Posterior dislocation

The Allis manoeuvre requires two practitioners. Assistant stabilises the pelvis by applying posterior pressure to pelvis. Operator stands on the stretcher. Gently flexes hip and knee to 90°. Hip is gently adducted and internally rotated while increasing sustained traction is applied by pulling on the knee. Be careful with rotational force – can cause iatrogenic femoral neck fracture. May hear of feel a ‘clunk’ on reduction.

The Whistler manoeuvre requires one practitioner. Patient is supine with both knees flexed and feet firmly on the bed. The arm is placed under the knee on the affected side and on the unaffected knee. The other hand grasps the affected leg just above or at the ankle. With the hip at 90° and slightly adducted, the arm in the popliteal fossa is used as a lever to apply traction to the hip by pulling down on the lower leg. Maintain a constant steady force. It may be necessary to place the practitioner’s leg on the bed with the knee in the popliteal fossa as fulcrum (Captain Morgan technique).

Anterior dislocation

Assistant applies longitudinal traction and gentle internal rotation to the femur in its abducted position. Practitioner holds the pelvis and applies gentle pressure to the femoral head to push it back into the acetabulum.

Repeat radiography is required to confirm reduction. Patients should be admitted to orthopaedics post-reduction or where reduction has failed.

Central dislocation

Central dislocations are rare. The femoral head is driven through the acetabular floor. All central dislocations require surgical reduction and repair.

210 Long-bone fractures of the leg

A large amount of force is required to fracture adult long bones. These injuries are usually associated with multiple other injuries. Femur fracture may cause massive blood loss into the thigh compartment that is difficult to detect on early exam.

The first five minutes

- ABC, IV, O_2 – control haemorrhage
- Check distal pulses, cap refill, and sensation
- Restore anatomic position and immobilise (repeat neurovascular assessment after immobilisation), analgesia

History and physical examination

Key historical features

- Evaluate mechanism and consider associated injuries
- Position of leg and direction of force applied
- Ability to walk after injury
- Pre-injury function, prior injuries or surgery

Signs and symptoms

- Look for swelling, deformity or open wounds
- Check distal pulses and sensation
- Repeat neurovascular assessment frequently, and evaluate for compartment syndrome (see 📖 Compartment syndrome, p. 566).

Investigations

- Labs: Hgb, type and cross match
- Imaging: XR (femur hb – femur, knee, AP pelvis; tibia hb–fibula hb – knee, ankle

Femur fractures

Types

- Subtrochanteric – proximal femur just below trochanters. In young patients associated with high velocity or high energy injuries (high risk of other injuries). In elderly, bone disease can lead to fracture from trivial force
- Femoral shaft – transverse, spiral or segmented. Often comminuted
- Supracondylar – distal third of the femur involving the femoral condyles and potentially the articular surface. May present with a knee haemarthrosis and ligament injury. High incidence of post-traumatic arthritis

Tibia-fibula fractures

- The tibia and fibula are often injured together
- Tibial plateau fractures – caused by femoral condyle driven into tibia
- Tibial shaft fractures – associated with crush injuries and pedestrian MVAs. Many are open. Associated soft tissue injuries may heal poorly and there is high incidence of compartment syndrome
- Isolated fibula fractures – often result from direct lateral blow. May be associated with lateral compartment syndrome or peroneal nerve injury

Management

The goal of acute management is to identify fractures, dislocations, and associated neurovascular injuries; to control bleeding and pain; and to immobilise to prevent further tissue damage.

- Early analgesia; consider regional nerve block (femoral nerve block or 3-in-1 block AFTER careful initial neuro exam and only if no risk for compartment syndrome)
- Immobilise the leg – Thomas splint or similar traction splint for femur fractures ◇; long leg splints either plaster, fixed or telescopic for tibia-fibula
- Tibial plateau fractures with > 3 mm depression require ORIF ◇
- External fixation for displaced or open tibia-fibula fractures ◇

- Blood transfusion as needed, early if ongoing blood loss ◇
- For open fractures, tetanus anti-toxin and cover with a first generation cephalosporin
- Serial exams for signs of vascular injury: Refer for angiography ◈ or immediate surgery ◇

Critical documentation

Serial VS. Full trauma survey. Serial neurovascular exams, before and after stabilisation.

Disposition

All open, displaced or severely angulated fractures and all femur fractures require immediate referral to orthopaedic surgery ◇

Admit patients in whom there is ANY suspicion of compartment syndrome. The fibula is largely non-weight bearing and isolated (without tibia or other ankle injury) non-displaced fractures can often be managed with casting. Displaced fractures are often associated with ligament disruption and may require surgical repair

Admit all poly-trauma patients with long-bone fractures.

211 Knee injuries

Knee injuries are very common and may be accompanied by significant vascular or neurological injuries. A good exam is the key to identifying dangerous injuries.

Key historical features

- Mechanism of injury
- Position of leg and direction of force applied
- Ability to walk after injury
- Pre-injury function, prior injuries or surgery.

Signs and symptoms

- Swelling (effusion), tenderness, deformity or open wounds
- Palpate along the joint line for tenderness
- Test active range of motion; if limited, test passive range of motion
- Evaluate stability of the ligaments and meniscal integrity (note that these tests may be limited in the setting of acute pain and oedema):
  - *Patellar apprehension test* (rapid extension of the knee with lateral force on the patellar). Quadriceps tightening is positive sign
  - *Lachman test* for anterior cruciate ligament (ACL). Knee is flexed to 20–30° and tibia pulled forward. Significant movement of the tibia anteriorly relative to the femur indicates ACL instability
  - *Posterior drawer test* for posterior cruciate (PCL). Knee flexed to 90°, tibia is moved posteriorly, compare the two sides, may be up to 5 mm of movement without ligament injury
  - *Laxity of medial collateral (MCL) and lateral collateral (LCL) ligaments* is tested by applying a medial or lateral force with the leg extended and knee flexed to 30° (isolates collaterals)
  - *McMurray test* for meniscal injury: Knee at 90°, apply medial or lateral force and compress menisci against femoral condyles. As leg is extended meniscal fragment may push out of the joint space

Investigations

The Ottawa knee rules suggest XR in patients with:
- Age > 55 years
- Isolated patellar tenderness
- Tenderness at head of fibula
- Inability to flex knee to 90°
- Inability to bear weight (four steps) immediately after injury and when examined

For suspected patellar injury: Lateral, AP, and sunrise views. US useful to assess for soft tissue injury and haemarthroses ◇.

Possible causes and differential diagnosis

Knee ligament injuries

- MCL most common. Occurs when valgus force is applied to knee. Injuries graded as Grade I – simple stretch, Grade II – partial tear, Grade III – complete tear. On examination: joint laxity, tenderness over medial joint margin. Effusions are rare
- ACL common in athletes. Occurs when valgus force is applied to the extended knee. A pop is often heard or felt. Initially there may not be limitation of movement but an effusion will develop with time. Look for associated segond fracture (avulsion of lateral tibial plateau) on X-ray
- PCL tears often occur together with other ligamentous or meniscal injuries. Occurs when posterior force is applied through a flexed knee. Effusions are rare
- LCL injuries are relatively uncommon in isolation. Result from a varus force to the knee

Knee dislocation is relatively rare but important. Critical to assess vascular status as is associated with high rate of vascular compromise – even with spontaneous relocation – which may lead to limb loss.

- Look for deformity, swelling, immobility. Many are reduced by the time of presentation and may not be obvious
- XR for associated fractures
- Assess fibular (peroneal) nerve injury (sensation in first webspace, impaired foot dorsiflexion)
- Careful vascular examination (compare with contralateral pulses)

The popliteal artery may be damaged in knee dislocation/subluxation. Immediate surgical revascularisation is indicated for signs of vascular injury ◈. In the absence of hard signs an ankle/brachial index should be performed. If less than 0.9, consult vascular surgery. Arteriography, CT angiogram ◈, or Doppler ◇, to evaluate vasculature where available.

Reduction is easily accomplished with longitudinal traction. Post reduction the knee should be splinted in 20° of flexion.

All knee dislocations should be admitted for serial perfusion checks.

Patellar dislocation

Patellar dislocation is caused by a lateral force applied to the flexed knee or excessive quadriceps contraction. Presents with knee locked in flexion, patellar felt laterally. Reduce by extending the knee and pushing patellar medially.

Patellar fracture

Caused by direct trauma, e.g. fall onto flexed knee. Fractures may be stellate, transverse or horizontal. If able to extend fully, give analgesia, immobilise and arrange follow-up. With limited extension, refer to orthopaedics for surgical repair ◇.

Quadriceps and patellar tendon rupture

Present with a swollen, tender knee with limited extension.

Quadriceps tendon rupture = low-riding patellar;
patellar tendon rupture = high-riding patellar.

Tibial plateau fractures

Result from lateral or medial forces applied to flexed knee. Lateral plateau fractures are more common. Present with a swollen knee, tenderness along joint margins and possibly an effusion. On X-ray a lipohaemarthrosis (layering of fat, blood and synovial fluid) may be seen. Depression of the tibial plateau may require fixation ◇. See 🕮 Long-bone fractures of the leg, p. 554.

Management and disposition

The goal of acute management is to identify fractures, dislocations, and associated neurovascular injuries; to control pain; and to immobilise to prevent further tissue damage.

- If ligament injury: splint or strap, non-weight bearing until follow-up
- If dislocated: Reduce urgently and immobilise, serial vascular assessments
- Immobilise soft tissue injuries in a bulky compression dressing (i.e. Robert-Jones, comprising alternating layers of cotton wool and crepe bandage), elastic support bandage or hinged splint (knee immobiliser) ◇
- Analgesia
- Excluding minor sprains, non-weight bearing until orthopaedic follow-up ◇
- Admit for neurovascular compromise and as needed for surgery

212 Ankle injuries

The talocrural joint (ankle joint) is the articulation between the tibia, fibula, and talus. Ankle injuries have the potential to cause significant loss of function.

The first five minutes

- Assess neurovascular status
- Immobilise ankle
- Provide analgesia

History and physical examination

Key historical features

- Mechanism of injury
- Ability to weight-bear since injury (unable → XR)

Signs and symptoms

- Palpate lateral and medial malleoli, and distal posterior tibia and fibula (tenderness → ankle XR)
- Assess base of 5th metatarsal and navicular (tenderness → foot XR)
- ALWAYS do complete knee examination in ankle injury
- Assess range of motion (active and passive)
- Check Achilles tendon intact

Ligament injuries

Lateral collateral ligament injury

Most common type of acute ankle sprain. Injury occurs through forceful inversion (adduction). Always check the medial side for fractures when there is a lateral ankle sprain or tear.

Medial collateral ligament injury

Typically occurs with forced eversion (abduction). Deltoid ligament tears occur concomitantly with medial malleolar fractures, fibular fractures, and sprains of the lateral ligaments or syndesmoses.

Tendon injuries

The Achilles tendon is the main plantar flexor of the foot, arising from the gastrocnemius and soleus muscles of the calf and inserting on the calcaneus. Sudden dorsiflexion may overstretch the tendon and cause rupture. Spontaneous

rupture may occur in older patients with underlying medical conditions (rheumatoid arthritis, gout, or hyperparathyroidism) or patients taking fluoroquinolones.

Immediate management consists of immobilisation in a position of plantar flexion ◇. Referral to orthopaedic (surgical versus non-surgical management).

Ankle fractures

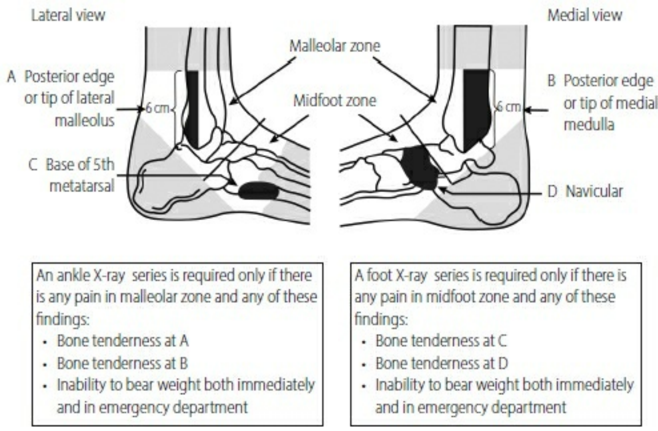

Figure 212.1 Ottawa ankle rules

Source: Brown & Wyatt. 2008. *Oxford American Handbook of Emergency Medicine*. By permission of Oxford University Press, USA.

Weber A

Supination-adduction or inversion injury – ankle is inverted, foot is supinated and talus adducted:
• The lateral ligaments tear or avulse a bony fragment from the lateral malleolus
• Oblique fracture of the medial malleolus at the level of the mortise
• Syndesmosis intact

Weber B

Supination-external rotation or eversion injury – talus is externally rotated, foot is either supinated or pronated:
• Transverse medial malleolar fracture and/or deltoid ligament tear
• Oblique fibular fracture at level of syndesmosis
• Partial syndesmosis tear

Weber C

External rotation injury:
• Deltoid ligament tear or avulsion fracture of medial malleolus
• Syndesmosis ruptured
• Spiral fibular fracture above the syndesmosis

Investigations

The Ottawa Ankle Rules help to determine which adult patients require XR.
Order **ankle** XR if there is:
• Inability to bear weight four steps at time of injury or when assessd
• Tenderness of lateral or medial malleoli
• Tenderness of distal 6 cm of posterior fibula or tibia.
In addition, order **foot** XR if there is:
• Pain in the midfoot area (from the level of proximal navicular through the proximal fifth metatarsal)
• Tenderness over navicular

- Tenderness at base of the fifth metatarsal

Management

- Analgesia
- If ligament injury – strap or elastic bandage for support, limited weightbearing until acute pain and swelling resolved, arrange follow up
- If fracture or dislocation: Reduce to neutral position, immobilise with below-knee splint, ankle in 90° dorsiflexion, non-weightbearing until follow-up
- Elevate leg
- Weber A fractures immobilisation for 4–6 weeks. Weber B and C fractures early orthopaedic follow-up ◇ – may need surgery ◇

Critical documentation

Serial neurovascular status, especially before and after reduction and immobilisation. Always document knee exam as associated injuries are common.

213 Soft tissue problems of the hand and foot

Untreated, soft tissue conditions of the hand and foot can result in substantial disability. The hand and foot contain many fascial planes and inflammation and scarring of connective tissue can result in functional compromise.

The first five minutes

- ABC, control haemorrhage
- Assess distal pulses, capillary refill and sensation
- Remove rings immediately if hand injury, as progressive oedema may rapidly compromise perfusion

History and physical examination

See also 🕮 Examination of the hand, p. 542.

- Mechanism and timing of injury
- Potential functional impact: hand dominance, occupation, social support
- Assess strength and sensation prior to local or regional anaesthesia
- Explore wound in position of injury to visualise tendon injury
- Nerve injury – test median, ulna and radial nerve function
- Tendon injury
- Test ability to flex and extend all joints against resistance
- Compare power to opposite hand

Infection

Bite wounds – human and animal

- High risk for infection
- Antibiotic prophylaxis, usually amoxicillin-clavulanic acid (clindamycin does NOT cover *Eikenella* species, important in human bites)
- Consult hand surgery if joint or tendon involvement or significant deformity ◈

Cellulitis, osteomyelitis, abscess, septic arthritis, and deep space infection

- CBC, ESR ◇, BCC ◈ if concern for osteomyelitis
- Broadspectrum antibiotics
- Elevation of extremity (‘sky hook’)

- Admit for serial exams. Hand surgery consult if not responding to antibiotics in first 24 hours or if drainage required

Flexor tenosynovitis

- Severe infection of flexor tendon sheath
- Pain along tendon, finger in flexion, sausagedigit swelling, pain with passive extension
- Surgical emergency: consult hand surgery early
- Splint and elevate hand, start broadspectrum antibiotics

Trauma

Digital and finger pad amputations

- Control haemorrhage
- Irrigate copiously to prevent infection
- Apply pressure, sterile and nonstick dressing; buddy tape
- If no bony or nail involvement, home with follow-up
- If bone exposure, hand surgery. May be necessary to rongeur and file bone tip to allow closure. Skin closure should not be under tension from bony extension
- If amputated digit and reimplantation surgical capacity available, cleanse amputated part with saline, wrap in gauze, place in watertight bag, then place bag in water with ice. Do NOT place digit directly on ice. Indications for reimplantation: thumb, multiple digits; most amputations in children; individual digit, amputation distal to insertion of flexor digitorum superficialis. Contraindications include crush injuries and amputations distal to the DIP joint

Nail bed laceration

- Highforce injury. May have associated subungual haematoma
- XR to rule out fracture ◇
- Small nail bed lacerations with intact nail and nail fold do NOT require nail removal and repair
- Only repair if uninfected and less than 24 hours old:
  - » Apply digital block
  - » Bluntly dissect away injured nail
  - » Close nail bed with surgical glue (some evidence for better outcomes) or small absorbable suture (6.0/7.0)
  - » Splint eponychial fold: place original nail fragment, sterile petroleum gauze, or sterile foil from suture wrapper in nail fold space to maintain opening and allow new nail growth

Subungual haematoma

- Painful collection of blood beneath nail. When haematoma > 50% of nail, laceration likely
- Subungual decompression:
  - » Drill multiple fullthickness holes into nail bed to drain haematoma
  - » Can use cautery unit ⟐, or paper clip or other sharp fine-tipped metal instrument heated to red hot for sterility

Distal phalanx fractures and dislocations

- Manage associated nail bed injuries
- Reduce dislocations with distal traction and hyperextension
- Closed fractures: reduce only if significant angulation/displacement
- Hand surgery if open, irreducible, or intraarticular fracture
- Splint and buddy taping for stability

Highpressure injection injury

- Highpressure gun injection of paint, chemicals, grease causes small entrance wound and severe deep tissue

necrosis despite benign appearance
- Surgical emergency
- If delay, consider debridement prior to OT
- Open midpalmar space, first web space for thumb, midlateral longitudinal incisions over involved fingers. Normal saline irrigation. Leave spaces open. Dress with saline-soaked sterile gauze

Tendon injuries

Extensor tendon phalangeal injuries

- Unable to extend against resistance
- PIP injury: central slip may become ‘boutonniere’ deformity
- DIP injury: mallet finger
- Consider associated avulsion fracture
- Posterior splint with MCP slightly flexed, DIP and PIP in full extension for 6–8 weeks
- Buddy tape

Flexor tendon injury

Jersey finger: injury of the 4th finger DIP
- Splint finger in comfortable position
- Hand surgery consultation ⟐

Ulnar collateral ligament injury (*game keeper’s thumb*)

- Forced abduction of the thumb
- Pain, swelling over ulnar aspect first MCP. Compare to uninjured side for ligamentous laxity on valgus stress, decreased pinch strength between first and second fingers
- XR to rule out avulsion fracture ◇
- Splint with thumb spica, MCP flexed at 20°. Complete thumb immobility for six weeks
- Early specialist referral if associated fracture, severe laxity ⟐

Foot

Plantar fasciitis

- Inflammation of plantar fascia, starting at calcaneal insertion
- Overuse injury
- Pain with first step out of bed or arising from chair; worse with walking
- Tenderness at heel pad and along arch of foot
- Management: NSAIDs, ice, rest, well-padded shoes

Ingrown toenail

- Management: nail excision under digital block
- Cut from tip of nail down to base, use forceps to pull out the nail
- Splint edge of remaining nail to prevent ingrowth
- Apply antibiotic ointment on sterile dressing
- Follow-up one week

Diabetic foot infection

- Poor wound healing, neuropathy predisposing factors
- Consider necrotising fasciitis if rapidly progressive, pain out of proportion, signs of septicaemia, or crepitus
- Antibiotic:

    - Inpatient: ceftriaxone and metronidazole
    - Outpatient: ciprofloxacin and metronidazole
- Very low threshold for admission for observation, twice daily wound checks and possible debridement
- Wound care for any wound, ulceration, or cellulitis

Critical documentation

- Medical conditions complicating wound healing (e.g. diabetes, malnutrition), tetanus status if open wound, follow-up plan
- Neurovascular status prior to local or regional anaesthesia, and before and after reduction or splinting
- In tendon injury: severity of injury and degree of function, interventions, follow-up

Disposition

- Referral to orthopaedics/hand surgery if persistent pain despite appropriate management, or if injury involves dominant hand and threatens patient's livelihood or ability to care for himself or others.

214 Compartment syndrome

Fascial membranes divide the upper and lower limbs into compartments. Elevated pressure within fascial compartments results in decreased perfusion, leading to tissue anoxia, swelling, necrosis, and nerve injury. Delayed treatment may result in deformity, disability, or amputation.

The first five minutes

- Assess pulses and sensation
- Analgesia
- Rapid orthopaedic consultation

History and physical examination

Key historical features

Any tense compartment or painful limb may represent compartment syndrome. Neurological findings and decreased pulses are late signs and usually indicate some degree of irreversible injury. Maintain a high degree of suspicion in order to make a timely diagnosis and intervene. Enquire about possible causes.

Signs and symptoms

Timing of symptoms may vary. Classic teaching of simultaneous pain, pallor, paraesthesia, paralysis is exceedingly rare. Stepwise progression of symptoms:
- Pain out of proportion to injury
- Persistent ache or deep burning pain
- Paraesthesias
- Pain with passive stretch of affected muscles
- Tense, firm compartment
- Decreased sensation or paraesthesias, suggesting nerve ischaemia
- Muscle weakness
- Paralysis (late finding)
- Pallor from arterial insufficiency (rare finding)
- Serial exams every 30–60 minutes. Rapid progression of symptoms and signs are strongly suggestive of acute compartment syndrome.

Possible causes and differential diagnosis

- Traumatic aetiologies:
  - Classically associated with closed long bone fractures, often tibia or distal radius
  - May also occur with open fractures, crush injury, burns, arterial or venous injury, severe muscle contusion or soft tissue injury, or post-operatively
- Nontraumatic aetiologies:
  - Extravasation of IV fluids, envenomations and bites, ischaemiareperfusion injury, thrombosis, prolonged limb compression following drug or alcohol intoxication or poor positioning during surgery, overly tight bandages or casts, tourniquets or circumferential constriction, minor trauma in those with bleeding diathesis
- Chronic exertional compartment syndrome may present as insidious pain in athletes
- Consider ischaemic limb, necrotising fasciitis in setting of pain out of proportion to exam
- Other aetiologies of limb oedema may contribute to compartment syndrome, including nephrotic syndrome, intra-arterial drug injection, severe deep venous thrombosis (phlegmasia cerulea dolens)

Investigations

- Labs: XR and crossmatch (bleeding disorder, OT) ◇; CK (rhabdomyolysis) ◈
- Compartment pressure measurement ◈

Management

Any delay in definitive management increases risk of limb amputation and death.

- Relieve all external pressure
  - Remove dressings, splints, casts, and any other obstructive object
  - Keep limb on level of the heart; do not elevate or make dependent
- Analgesia
- IVF for hypotension
- Broadspectrum antibiotics if suspect complicating infection or if prolonged muscle damage
- Grampositive antibiotic coverage for surgical prophylaxis at fasciotomy
- If myoglobinuria due to rhabdomyolysis, aggressive IV hydration (See 📖 Rhabdomyolysis p. 617)
- Fasciotomy to decompress compartments ◇
- Recommendations for pressure level at which fasciotomy is indicated vary

When in doubt, compartment should be released with fasciotomy.

Critical documentation

- Suspected cause, serial exams and progression of symptoms, and compartment pressures, if measured. Impact of interventions. Medications given.

Disposition

- Fasciotomy must be performed urgently for best outcome. Longer delays result in worse outcomes. Irreversible muscle and nerve damage begin at four hours
- If orthopaedic consultation or immediate transfer to higher level of care is not possible, consider stabilisation with emergency fasciotomy, followed by transfer for definitive management

215 Back pain

About 90% of adults will experience back pain at some time. Most episodes resolve within six weeks and with no specific diagnosis. The aim of management is to identify signs of potentially serious pathology – ‘red flags’.

The first five minutes

Focus on identifying serious underlying conditions:

- Is there neurological compromise requiring specialist management?

• Is there an underlying systemic disease such as infection or neoplasm?

Key historical features

Careful history should include:
• Age (if ≥ 50 or ≥ 20, consider infection or cancer)
• Duration of symptoms and response to previous treatment
• Intensity of pain. If not relieved at rest or worse when supine – consider infection or neoplastic disease
• Any urinary changes (urinary retention may present as new or frequent night-time urination)
• Fever
• Pain radiating below the knee
• Unexplained weight loss
• History of chronic infection including skin furuncles
• History of neoplastic disease – especially prostrate, breast, lung, and kidney (prone to skeletal metastasis)
• History of trauma – significant trauma in the young but minor falls, heavy lifting or severe coughing bouts in the older patient
• History suggestive of neurological compromise, i.e. tingling (paraesthesia), altered sensation and motor or sphincter dysfunction as in cauda equina syndrome

Signs and symptoms

The physical examination may point to serious conditions such as neoplastic or infective disease.
• Fever, vertebral tenderness and very limited range of movement may suggest spinal infection
• Neurological examination should include:
  » Straight leg raise to test for sciatic symptoms
  » Check power of the lower limbs (especially L4 – ankle dorsiflexion, L5 – big toe extension)
  » Increased reflexes suggest UMN pathology (spinal cord); decreased or absent reflexes suggest LMN (peripheral nerve root)
  » Sensation will be diminished in a dermatomal pattern – e.g. L4 – medial aspect of leg and ankle, L5 – lateral aspect of leg and dorsum of foot; S1– lateral aspect of foot (see Figure 215.1)
• Urinary changes can be associated with spinal compression at any level

Investigations

Spinal XR ◇: limit to elderly patients, and those with features suggestive of trauma or systemic disease (infection, malignancy, etc.). Low threshold for XR in elderly patients, in whom compression fractures can result from minimal trauma (e.g. coughing). AP and lateral views are adequate.

CT Scan ◈: for patients with pain > 6 weeks or high-risk features – shows excellent bony detail and may be adequate for disc pathology and paraspinal tumour or abscess, although less sensitive than MRI.

MRI ◈: investigation of choice for most spinal pathology. Emergency indications are cauda equina syndrome and any progressive neurological deterioration. MRI can be used to evaluate the cord, cauda equina and nerve roots and also tissue outside the spinal canal. It does not visualise bone well. Some patients may have difficulty lying still for the examination.

Spinal myelography ◈: has largely been replaced by MRI. Using dural injection of water-soluble contrast, most intra-spinal pathology can be detected. Combined with CT, better visualisation of the spinal cord and nerve roots is achieved, although it may miss extradural pathology. Can be used to evaluate the cord and cauda equine and provides very accurate information about the degree of spinal narrowing. However, it is invasive and carries a small risk of infection and post LP headache, nausea and vomiting.

Management

In patients with no red flags and no specific diagnosis after initial assessment, conservative treatment for up to six weeks.

Non-surgical conservative treatment

In the absence of clinical features suggestive of serious spinal pathology, **activity modification**:

- Rest: most patients do not require bed rest. If needed, should not be for more than 2–4 days. Limit heavy exertion, heavy lifting, prolonged sitting and bending or twisting the back to help reduce symptoms. Minimal daily activity within tolerable level of discomfort should be continued
- Exercise: may include walking, bicycling or swimming. A programme of physiotherapy ◇ is helpful if symptoms persist. Patient education with regards to posture, sleeping positions, lifting and bending techniques
- Analgesics: short-term analgesics such as paracetamol ± codeine. Consider other opiates only for severe pain
- Muscle relaxants: small doses of diazepam may be effective
- Other medications: for chronic treatment of neuropathic radicular pain, consider pregabalin or gabapentin
- Physical treatments: ice and heat may be beneficial in acute back pain. Lumbar support belts and corsets have no proven benefit for acute back pain
- Spinal manipulation ◇: done by trained practitioner, may be helpful for patients with acute back pain in the first month of symptoms. Should not be used in patients with neurological features
- Epidural injections ⟐: steroid injections may be beneficial for short-term relief of radicular pain. Risk of infection
- Injection treatment (◇ and ⟐): acupuncture may be beneficial in chronic but not acute back pain

Surgical treatment ⟐

Indications may include:

- Acute presentation: severe or progressive neurological deficit as a result of compression from trauma, infection, neoplasm or degenerative disc disease.
- Subacute presentation: compressive disc lesions with failed response to conservative management
- Chronic: most will have spinal spondylotic disease with varying degrees of stenosis and spondylolisthesis

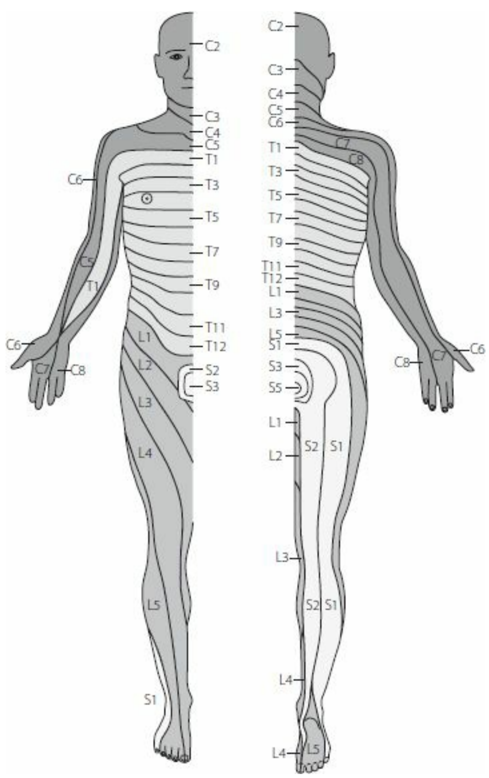

Figure 215.1 Dermatomes

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Part

4

N. Psychiatry

216 Medical clearance of mental health patients

Psychiatric symptoms will have a medical cause only in a minority of patients presenting for acute care; however, these patients may also have difficulty communicating regarding symptoms and must undergo a full history and physical examination. The rate of clinically important medical conditions in patients presenting to acute care settings for psychiatric symptoms varies greatly by region, and is substantial in some parts of Africa. The most common reason overall for missing a medical cause for a mental health presentation is failure to take an adequate history and physical, and failure to perform a mini mental state examination.

Differential diagnosis

Overdose

- May be asymptomatic; consider paracetamol ingestion in all patients
- Look for specific toxidromes; consider polydrug if clinical findings are mixed

Psychotic disorders

- Delirium (including secondary to HIV, metabolic and acute CNS infections, substance use/withdrawl)
- Drug intoxication
- Hypoglycaemia
- Post ictal state, ongoing seizure

Mood disorders

- Thyroid disease (hyper- or hypothyroid)
- Substance use/withdrawal

Investigation

First presentation of mental health problem

Do a thorough clinical assessment with history, serial VS (especially if any presenting VS are abnormal), complete physical examination, blood glucose, urinalysis in all patients, and pregnancy test in all women 12–50 years. If any

of these are abnormal, then further investigation as clinically indicated.

Literature on the subject is variable, but patients over 40 and those with known or suspected immunocompromise should undergo laboratory testing and brain imaging. In general, presentations of psychosis will require the most workup.

Consider:
- **Renal function** and electrolytes if dehydrated/delirious/or relevant to current medications
- **Thyroid function** if mood disorder/unexplained tachycardia/significant symptoms of thyroid disease
- **Toxicology testing**. Low threshold for paracetamol testing if suspected or reported ingestion. Screening for drugs of abuse where available
- **HIV testing** if any clinical signs of immunocompromise
- **Lumbar puncture** if clinical signs of immunocompromise, atypical age for first psychotic presentation, or absence of risk factors for psychotic disease
- **Syphilis serology** if elderly and new psychotic symptoms
- **CT** for recent history of head trauma, immunocompromise, age > 40

Known mental health disorder

Any patient with a known mental health disorder, but presenting with a relapse of prior symptoms while on treatment:
- Do a thorough clinical assessment with VS, O_2 sat, urinalysis, blood glucose, and pregnancy test
- Do serum drug levels when relevant (e.g. lithium). If these are normal and no clinical suggestion of a new medical problem, further testing is not indicated
- If symptoms are different from prior, workup as new onset

High risk population

A lower threshold for investigations should be considered in patients with an increased likelihood of co-morbid medical conditions and medical causes of their mental health symptoms:
- Elderly
- Alcohol and other substance abusers
- Immunosuppressed

Management

The goal of acute management is to:
- Detect medical causes contributing to mental health symptoms
- Detect comorbid medical conditions requiring acute treatment

217 Mental state assessment

This involves a systematic approach to evaluating and recording a patient's mental functioning. The aim is to diagnose probable cause of illness and guide treatment options. In the acute setting it should be brief and focused.

In agitated, withdrawn or uncooperative patients, observation and collateral information are critical. Introduce yourself, establish a rapport and conduct the interview in a quiet, safe environment. Ask open-ended questions. Allow the patient to answer in their own words, and encourage elaboration and explanation. Avoid interrupting. Listen and observe closely.

Appearance

- Personal hygiene – is there evidence of self-neglect?
- Is the dress appropriate for the weather and surroundings?
- Is the patient dressed elaborately?
- Are there any abnormal movements (posturing, tics, grimaces)?

Behaviour

• Is the behaviour socially appropriate for the setting?
• How does the patient relate to the examiner?
• Does the patient maintain eye contact?
• Does the patient cooperate?
• Is the patient easily distracted, restless, aggressive?
• Is the patient quiet and withdrawn?

Speech

Relates to speech characteristics, not content.
• Assess volume, rate and tone
• Are answers brief and monosyllabic or inappropriately long?
• Is the speech pressured, loud or mumbled?
• Note dysarthria, aphasia

Emotional state

The patient describes the subjective component. Also observe for emotions conveyed through body posture, facial expression and vocal tone.
• Is the mood elevated or depressed?
• Does the patient appear restricted or flat?
• Is the mood changeable in range or intensity?
• Are there anxiety or panic symptoms?
• Is the mood appropriate to the content of the discussion?
• Ask about concentration span, appetite, and feelings of guilt, worry, sleeping patterns and sexual performance

Thought disturbance

Thought form refers to how the patient links ideas.
• Are the answers appropriate to the content of the conversation?
• Does the conversation follow logically?

Formal thought disorders

• Circumstantiality: over inclusion of irrelevant details
• Clang association: association of thoughts occurs by sound rather than meaning (e.g. ‘I sleep well’, ‘I peep well’)
• Derailment or loose associations: illogical connection between ideas and sentences do not make sense
• Flight of ideas: moves rapidly from idea to idea
• Neologism: invented words or using conventional words in incorrect ways
• Perseveration: repetition of words, phrases or ideas, often out of context
• Tangentiality: answers a question with an answer related to the topic but doesn’t answer the actual question
• Thought blocking: thought or the flow of ideas is suddenly broken

Thought content refers to the ideas conveyed.
• Probe for suicidal or homicidal thoughts (ask directly)
• Delusions are illogical or false beliefs, held rigidly despite evidence against them. They include paranoid, grandiose, religious or somatic beliefs
• Obsessions are intrusive, uncontrollable thoughts, often embarrassing or hurtful (e.g. wanting to shout obscenities)
• Preoccupations dominate the person’s thinking (e.g. worry about finances)
• Phobias are irrational, excessive fears about specific things or situations

Perceptual disorders

Hallucinations

Sensory perceptions in the absence of external stimulus (e.g. hearing voices in one's head). They may be auditory, visual, tactile, olfactory or gustatory.

Illusions

Distortion of reality resulting from misinterpretation of true sensory stimuli (e.g. rustling curtain misinterpreted as a snake). More common in delirium.

Cognition

Describes overall function of the CNS. See 📖 Mini Mental State Examination p. 938.

Insight

Capacity to understand one's own illness and need for treatment.

218 Risk assessment in suicidal, homicidal and violent patients

The majority of suicide attempts are a once off event, but 16% repeat within a year. Prior suicide attempt is the best predictor for future attempts, with 10% eventually succeeding. Patient report of a profound feeling of hopelessness is the strongest predictor of eventual suicide.

Interviewing

- Complete assessment when patient is not intoxicated, establish rapport, and corroborate with family/friends
- Assess intent. Observed affect and behaviour may increase suspicion even if intention is denied
- Identify triggers and predisposing factors
- Determine access to means of suicide or harm to others
- Assess mental state

Asking questions about intention is difficult but essential. Asking about suicide does NOT prompt patients to consider suicide.

- Ask directly: Are you feeling hopeless about the present or future?
- If yes, ask – Have you had thoughts about hurting yourself or hurting someone else? About taking your own life? Do you think about hurting any specific people or people in general? Do you ever hear voices or have intrusive thoughts that tell you to hurt yourself or others?
- If yes, ask – When did you have these thoughts? Do you have a plan? Have you ever hurt someone else or attempted suicide?

Higher risk for homicide

- History or increasing incidence of violent behaviour or arson
- History of stalking, hostage-taking or abduction
- Perpetrator idolises, expresses ownership of, or isolates target from others
- Access to weapons or history of choking or strangling victim
- Specific plans of violence expressed
- Frequent use of alcohol or drugs
- Response to internal stimuli

Lower risk for suicide

- Few significant risk factors (low SAD PERSONS score)
- Stable, supportive home/supportive person staying with patient
- Agrees to return if situation worsens
- Reliable health care access

- Specific follow-up within 48 hours
- Young female with nonlethal ingestion/‘hesitation cuts’
- Strong wish to live

Modified SAD PERSONS score for high suicide risk

This is a suicide risk stratification and management guide; it is not strong enough alone for discharge, but identifies the high risk patient.

S = Sex (male)
A = Age (< 19 or > 45)
D = Admits to depressive symptoms *
P = Previous suicide attempt or previous psychiatric care
E = Excessive alcohol or drug use
R = Rational thinking loss *
S = Single, separated, divorced or widowed
O = Organised or serious suicide attempt *
N = No social support
S = Stated future intent *

* Score 2 points if affirmative. All other questions score 1.

SCORE
≤ 5 Low risk = Potential discharge
6–8 Moderate risk = Psychiatric consultation
≥ 9 High risk = Likely admission

Follow up

- Discuss stressors and offer help-seeking and problem-solving strategies
- Plan appropriate outpatient appointments
- Institute a safety plan. Elicit a commitment not to reattempt suicide and to return for help if needed (‘contract for safety’)
- Consider holding patient for a cooling off period if concern for violence. Document clearly and know regionally-specific legal reporting requirements for threats of violence against specific individuals. Independent of legal requirements, every precaution should be taken to protect specifically threatened individuals, while maintaining patient confidentiality as best possible

Discharge criteria

- Medically stable; no intoxication or delirium
- No near-term suicide or homicide risk
- Lethal means of self-harm or violence removed
- Plan in place to return if any suicidal or violent intent
- Treatment of underlying psychiatric problem arranged
- Identification of precipitating crisis, resolution plan identified
- Physician confident patient will follow-up; social supports concur with discharge

219 Self-harm

The first five minutes

- VS, ABC
- Evaluate the need for immediate intervention for toxicologic and traumatic injuries
- Ensure safety of staff and remove any weapons/toxins from patient

• Reassure patient and provide direct observation in a safe environment to prevent further harm

History and physical examination

Key historical features

• Extent of injuries/ingestions
• Prior suicidal thoughts or attempts
• Ongoing intent for self-harm
• Specific plan for self-harm

Signs and symptoms

• Examine for evidence of injury, intoxication, or acute medical condition
• Look for specific toxidromes

Differential diagnosis

Non-suicidal self-harm

This includes self cutting, burning, scratching, hitting, hair-pulling
• Usually an attempt to convey feelings, relieve stress or soothe patient
• More common in western cultures
• Only a small proportion have active suicidality
• While usually not intended as suicide, self-harm behaviours may nonetheless be life-threatening

Suicide attempts

As in many areas, our understanding of the epidemiology of suicidal behaviour in Africa is limited by lack of data and cultural norms that inhibit open communication about suicidality. Worldwide, suicide is more common in young adults. In southern and Central Africa, there is an association with female gender, lower socioeconomic state, and White and Indian ethnicity. In eastern and western Africa, gender is not associated with suicidality, but lower educational level is.
Stressors:
• Relationship difficulties
• Socio-economic deprivation
• Loneliness
• Mental illness – including adjustment disorder and acute stress reaction

Common methods of attempt:
• Organophosphate pesticides, rodenticides, pills (antimalarials), other household poisons

Investigations

• Investigations based upon presenting complaints and physical findings
• Pregnancy testing for all women of child bearing age
• Medically clear the patient (📖 p. 574)

Specifically consider:
• Tests to aid management of poisonings (📖 p. 652)
• XR to identify foreign bodies in wounds or fractures in hanging attempts

Management

The goal of acute management is to limit complications associated with self-harm injuries, mitigate further harm, and medically clear patient to facilitate psychiatric evaluation.

Treat poisonings and any injuries appropriately (including ensuring up to date tetanus prophylaxis for all skin wounds).

- Ensure a safe environment and prevent patient from leaving before assessment is complete
- Undertake risk assessment (📖 p. 578)
- Maintain suicide precautions in high-risk patients

Critical documentation

- Note acute suicide risk, social circumstances and follow-up plan
- Clearly document reasoning when holding patient for urgent psychiatric evaluation
- Complete mental health documentation according to local laws
- Careful documentation of indications if restraints used

Disposition

- Risk related (📖 p. 578).
- Refer if appropriate testing and monitoring not available

220 The agitated patient

Agitated behaviour is a frequent emergency presentation, and includes violence, mood changes, disorientation, disturbed thinking, anger and changes in level of consciousness. The critical step is to distinguish general medical conditions from functional or psychiatric conditions.

The first five minutes

- VS, ABC
- Ensure safety of the patient and others; initiate early containment (may require chemical and/or physical restraint)

History and physical examination

A good history, especially from collateral sources, is essential. Complete the Mental Status Examination (📖 p. 576). Maintain a high suspicion for delirium or other organic disease. Complete a thorough physical examination (preferably before sedation), including blood glucose, pulse oximetry and urinalysis ◇.

Differential diagnosis

Agitated behaviour may occur in the context of physical or psychiatric illness, substance abuse or personality disorder.

Delirium

- An acute confusional state caused by organic brain disturbance
- Key features: disturbance of consciousness (agitation or drowsiness); cognitive disturbance (memory, orientation, attention, speech); usually acute onset with a fluctuating course; more common in the elderly; hallucinations are classically non-auditory
- These patients should have a thorough investigation for an organic cause

Mnemonic for differential diagnosis of delirium: DIMTOPS

- Drugs – intoxication, withdrawal or poisoning
- Infection – meningitis/encephalitis or other, e.g. UTI, pneumonia
- Metabolic disturbance – electrolyte or endocrine
- Trauma – head trauma

• Oxygen – hypoxia
• Postictal state
• Space occupying lesion (intracranial)

Dementia

• An organic brain disturbance with progressive decline in intellectual functioning, and change in behaviour and personality
• May present with a delirium on the background of progressive decline

Drug intoxication or withdrawal

• Manage as a medical emergency
• Drugs may be illicit or prescribed; substance abuse may accompany psychiatric illness
• Intoxication is an abnormal neurologic state caused by administration of a psychoactive substance:
  » Key features: agitation or decreased LoC, impaired judgement, impaired motor coordination
• Withdrawal is a clinical syndrome induced by decrease or cessation of a drug:
  » Agitation may be the presenting symptom. Other symptoms depend on the substance involved

Psychosis

• A dysfunction in capacity for thought and processing of information
• Feature of schizophrenia, mania, acute stress reactions, depression with psychotic features or personality disorder
• Key features: delusions, hallucinations, disorganised speech, disorganised behaviour, negative symptoms
• Acutely psychotic patients are not usually disoriented

Mania

• A manic episode presents as an excessive persistently elevated mood
• Consider organic causes, especially medications
• Key features: grandiosity or inflated self-esteem, increased talkativeness and physical activity, agitation, distractibility and impulsivity
• Associated psychosis can occur

Mood disorders

Abnormal mood states include depression, anxiety and acute stress reactions. Any of these may present with agitation or psychosis. Be alert for suicidal intention.

Personality disorders

Personality traits that are inflexible and maladaptive, may present with aggression or psychosis. Consider borderline, narcissistic and antisocial personality disorders.

Investigations

Focus workup to exclude a general medical condition (see 🕮 Medical clearance of mental health patient, p. 574).

Management

The goal of acute management is to control agitation in order to keep patient and staff safe, reduce patient discomfort, and facilitate appropriate aetiologic evaluation.

Ensure safety

• Keep calm and work in a team

- Do not confront the patient without adequate help (security/police)
- Remove weapons
- Evaluate the patient in a safe room with a ready route of escape: ensure the patient is not between you and the door
- Avoid making the patient feel threatened. Do not sit too close. Speak calmly and reassuringly
- Patients may become calm when confronted with a show of force. Approach as a group

Non-pharmacological

- Talk the patient down calmly, softly, and sympathetically. Continuously explain what is happening. Do not confront or threaten
- Ensure a calm, quiet area
- Use physical restraints for the briefest possible time until medications take effect
- Ensure adequate staff. Inform the patient that they will be restrained. Encourage co-operation
- Use five-point immobilisation:
  - One person per limb and one for the head
  - Grasp all extremities at the same time
  - Place the patient supine on the bed
  - Apply restraints to each ankle and wrist, and attach to bedframe, not rail
  - Ensure restraints are soft – leather preferred
  - DO NOT apply over neck, chest, head or use gags
- Seclusion should be done in consultation with a senior in an area free of hazards and visible to staff at all times. Do not seclude sedated patients

Rapid tranquilisation

VS should be monitored every 15 minutes until patient is ambulatory.

Oral treatment:

- If on a regular antipsychotic, lorazepam 1–4 mg
- If no regular antipsychotic, haloperidol 2.5–5 mg or risperidone 1–2 mg
- Repeat after 45–60 minutes. If two doses fail, go to step 2

IM treatment:

- Lorazepam 2–4 mg (have flumazenil available for respiratory depression. Caution – paradoxical disinhibition)
- Midazolam 7.5–10 mg
- Promethazine 50 mg (slow onset. Allow 1–2 hr for response. Max 100 mg per 24 hours)
- Haloperidol 5–10 mg should be last drug considered. High incidence of acute dystonia. (Treat dystonia with biperidin 5 mg IM or procyclidine 5 mg IM, or promethazine 50 mg IM)

IV treatment:

- Monitor CLOSELY and be ready to treat hypoventilation and hypotension
- Diazepam: start with 5 mg over 5 min, may repeat 5 mg after 10 minutes, max 40 mg per 24 hours (long-acting medication: CAUTION with stacked doses) OR
- Lorazepam 2 mg. Maximum 8 mg in 24 hours OR
- Haloperidol 2.5–10 mg IV over 5 min, may repeat after 10 min. Max 20 mg per 24 hours (but see comments above)

Critical documentation

Serial VS. Document any physical or chemical restraint needed. Document all interventions and clinical response. Complete locally required medico-legal documentation.

Disposition

- Agitated patients should be moved urgently to a calm and safe environment with adequate security

- Discharge patients who remain calm, and are medically well and appropriate for outpatient psychiatric management where available. Admit if illness or injury requiring admission; admit to Psychiatry if a risk to self or others, persistent agitation, or previous failed outpatient management

221 Depression

Depressive disorders are mood or affective disorders which usually occur as exacerbations between periods of normal function. Major depression is a persistent sad or depressed mood, or pervasive loss of interest in usual activities, lasting for at least two weeks.

By 2020, depression is projected to become the world's second greatest cause of loss of productive life-years. It often coexists with other psychiatric illnesses, and is frequently overlooked in the elderly. The lifetime risk of suicide in majorly depressed patients is 15%.

The emergency clinician must evaluate a patient's need for both medical and psychiatric intervention.

History and physical examination

Key historical features

Evaluate suicidality/homicidality (📖 p. 578). Ask explicitly about any ingestions or attempts to harm self. Any recent stressors or changes in job, family, housing. Any current or prior substance use psychiatric medication use (any missed doses?). History of psychiatric hospitalisations or illness. Obtain history from family and friends as possible/needed. Assess risk factors for major depression, which include: female sex, family history of depression or suicide, and other medical or psychiatric illnesses in the patient.

Signs and symptoms

Review VS for potential toxidromes, careful examination for evidence of trauma, endocrine anomaly or other acute medical problem.

Psychiatric examination should note general appearance, eye contact, speech and motor activity. Depressed patients often avoid eye contact and have a blunted affect. Characteristic symptoms are listed below. Onset may be sudden or gradual but most patients present to care after several weeks of symptoms.

SIG: ESCAPE: Mnemonic for depressive symptoms

Sleep (poor)	Energy (decreased)
Interest (lacking)	Suicidality
Guilt	Concentration (poor)
	Appetite
	Psychomotor (retardation or agitation)
	Emotion (depressed mood)

Differential diagnosis

Medical causes of AMS include drugs, thyroid disease, tumour syndromes and infections.

Consider also dysthymia, stress reactions (bereavement, etc.), adjustment disorder with depressed mood, bipolar disorder.

Investigations

See 📖 Medical clearance of mental health patients, p. 574.

Management

The goal of acute management is identification and treatment of medical causes, relief of symptoms and evaluation

of risk for harm of self or others.
• Provide safe environment and observation for all patients at risk of self-harm
• If high risk, arrange emergency psychiatric consult. Utilise social work services as appropriate
• Utilise benzodiazepines in anxious patients
Typically, acute care clinicians do not initiate antidepressant medications; however, it may be appropriate to prescribe a small amount in consultation with a psychiatrist.

Critical documentation

Note acute risk of harm to self or others, social circumstances and follow-up plan. Clearly document reasoning in writing when holding patients for urgent psychiatric evaluation. Complete medico-legal documentation according to local laws.

Disposition

• Discharge home if low-risk: arrange outpatient psychiatric follow-up and discuss with patient's primary doctor and family when possible
• Emergency psychiatric consultation if risk of harm to self or others

222 Anxiety

Anxiety disorders result from a combination of biopsychosocial factors, including genetic vulnerability and situational elements. Patients may present for acute care with feelings of severe anxiety or panic, depressive symptoms, or suicidal ideation. Anxiety may be a symptom of organic disease.

The first five minutes

• Evaluate the patient in a quiet area and give reassurance
• ABC, VS, consider medical causes for any abnormal VS
• Consider early sedative medications for severe attacks

History and physical examination

Key historical features

Onset of symptoms, triggering factors, prior episodes; ask about medications, drugs (amphetamines, cocaine, caffeine, energy drinks, nicotine, marijuana), risk factors for cardiac disease and PE, and psychiatric history. Consider accidental exposure in contaminated products (i.e. marijuana laced with amphetamines).

Signs and symptoms

Autonomic arousal resulting from anxiety may present with any of the following:
• Respiratory: hyperventilation, dyspnoea
• Cardiovascular: palpitations, chest pain, sweating
• Gastrointestinal: dry mouth, dysphagia, nausea, epigastric pain, loose stools
• Neuromuscular: tremor, headache, dizziness, tinnitus
• Genitourinary: urinary frequency, amenorrhoea, menstrual discomfort, impotence
• Hypocarbia: perioral paraesthesia, numbness, tingling, carpopedal spasm
• Psychiatric: 'sense of impending doom', depersonalisation, paranoia, phobic behaviour, agitation

Differential diagnosis

Psychiatric categories of anxiety disorders:
• Anxiety due to a generalised medical condition
• Substance-induced anxiety disorder
• Generalised anxiety disorder

• Panic disorder
• Adjustment disorder with anxious features
• Obsessive-compulsive disorder
• Phobias

Medical:
• Acute coronary syndrome
• Dysrhythmia
• Bronchospasm
• PE
• Hyperthyroidism
• Pheochromocytoma
• Hypoglycaemia
• Drug intoxication or withdrawal

Factors that point towards organic cause

• Onset of anxiety symptoms after 35 years of age
• No family or personal history of anxiety
• No childhood history of anxiety
• Lack of avoidance behaviour
• No acute stressor
• Poor response to anxiolytics

Investigations

• Labs: BG; electrolytes, ABG ◇; cardiac enzymes, TSH ◈
• ECG ◇
• Imaging; CXR ◇

Management

The goal of acute management is to relieve significant psychological, respiratory or circulatory symptoms and to limit recurrent events.
• Ensure calm environment; reassure
• Consider IM/IV anxiolytics

Critical documentation

Serial VS. All interventions and clinical response. Screen for suicidal and homicidal thoughts. Complete required medicolegal documentation according to local regulations.

Disposition

See 🕮 Medical clearance of mental health patients, p. 574.
• Admit to medical ward when indicated
• Admit to psychiatry if patient poses a risk to self or others, or for failure of outpatient management or social problems
• Discharge if controlled medical cause with medical emergency ruled out

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Part

4

O. Renal and urology

223 Approach to decreased urine output

At least 0.3 ml/kg/hour over 24 hours (approx 500 ml/day in adults) of urine is needed to clear the body of solute. Less than this is 'oliguria': causes include hypovolaemia, obstruction, neurogenic bladder dysfunction, toxicologic exposures, and acute or chronic kidney injury (see 🕮 Acute kidney injury, p. 595 and Chronic kidney disease, p. 596).

The first five minutes

- ABC, VS, O_2, IV (consider fluid challenge if not acutely hypoxic or dyspnoeic), cardiac monitor
- ECG; look for changes of hyperkalaemia
- Catheterise bladder
- Look for associated life-threatening conditions

History and physical examination

Key historical features

- Volume loss: decreased oral intake, vomiting or diarrhoea (± blood), physical exertion, recent or current infection, pyrexia, haematuria, trauma
- Obstruction: difficulty with micturition, hesitancy, dribbling, incomplete voiding, prostatism, pelvic pain, urogenital prolapse, constipation
- Toxicologic: anti-cholinergics or nephrotoxics
- Past medical history (hypertension, diabetes, cardiac failure, renal failure, neurologic disease, cervical or other malignancy)
- Past urogenital history (urethritis, trauma, stones, malignancies)
- Past surgical history (abdominal, pelvic, neurologic)

- Any recent travel history

Signs and symptoms

- Hydration status: capillary refill time, skin turgor, peripheral temperature, subcutaneous oedema, US of IVC (for intravascular volume)
- Pulmonary oedema, dyspnoea: volume overload (consider primary renal failure, or CCF causing AKI via poor cardiac output)
- Cardiac disease (hypertrophy, dilation, murmurs)
- Abdominal examination (palpable distended bladder, abdomino-pelvic masses, enlarged prostate, tender prostate, faecal impaction)
- Neurologic deficit (lower extremity strength and peri-anal sensation)

Possible causes and differential diagnosis

Pre-renal (abnormal renal perfusion)

- Hypovolaemia (dehydration, blood loss, burns), sepsis, poor cardiac output

Renal (intrinsic renal disease)

- Acute tubular necrosis (myoglobin / uric acid / calcium / transfusion / drugs / toxins / sepsis)
- Glomerular disease (diabetes/drugs/immune mediated)
- Reno vascular disease (renal artery stenosis/renocortical ischaemia/renal emboli/renal vein thrombosis, etc.)
- Interstitial disease (interstitial nephritis/diabetic nephropathy/hypertensive nephropathy/HIV nephropathy)

Post-renal

- Prostatic hypertrophy
- Urologic tumours/stones/masses
- Urethral strictures/valves/phimosis/paraphimosis/trauma
- Neuropathic bladder
- Intraperitoneal mass (cervical cancer)
- Faecal impaction

Investigations

- Labs: electrolytes, renal; urinalysis (proteinuria suggests glomerular disease), urine microscopy for cells, casts and cellular debris ◇; urine sodium and creatinine ◈
- ECG ◇: (signs of hyperkalaemia)
- Imaging: US (bladder volume ≥ 1 l suggests chronic obstruction with bladder dilation ◇), kidney, ureter, bladder XR ◇

Management

The goal of acute management is identification and correction of the underlying aetiology. Catheterise the bladder; determine post void residual by US.

Pre-renal

Optimisation of cardiac output and intravascular volume status.

Renal

Consider renal replacement therapy if renal failure.

Post-renal

Relieve urethral obstruction – urology for ureteric stent.

Critical documentation

Serial VS, response to therapy, strict fluid balance (input and output).

Disposition

Admit according to the underlying condition.

224 Acute kidney injury

Acute kidney injury (AKI) has replaced the term acute renal failure. It is a reversible rapid decrease in kidney functioning resulting in a failure to maintain fluid, electrolyte and acid-base balance. Patients may have severe extra-vascular volume overload (causing pulmonary and tissue oedema) with intravascular depletion (causing poor perfusion).

Prompt identification and appropriate management of AKI will minimise the risk of the patient developing irreversible damage.

The first five minutes

- ABC, VS, O_2, IV, pulse oximeter, cardiac monitor
- Evaluate for pulmonary oedema and hyperkalaemia

History and physical examination

Key historical features

- Urine output (number of wet nappies in small children)
- Fluid loss (vomiting and diarrhoea)
- Recent febrile illness (particularly streptococcal)
- Muscle injury (rhabdomyolysis)
- Medications

Signs and symptoms

- Weakness (hyperkalaemia)
- Dyspnoea, crepitations, cough (pulmonary oedema)
- Lower abdominal pain, distension (if bladder obstructed)
- Assess volume status – hypovolaemic, euvolaemic or hypervolaemic

Possible causes and differential diagnosis

Possible causes

Type	Cause	Urea: Creatinine ratio
Pre-renal	Poor renal perfusion: trauma, dehydration, burns, septic or other shock, DKA, renal artery stenosis/ thrombosis Children: diarrhoea	> 20
Renal	Nephrotoxins (including medications), glomerulonephritis, renal vein thrombosis, acute tubular necrosis, acute interstitial nephritis.	10–15

	Children: post-strep glomerulonephritis, tumours	
Post-renal (obstructive)	Ureteric stones, tumours, prostatic hypertrophy, intraperitoneal mass (cervical CA)	Normal

Differential diagnosis

• CCF (consider secondary renal failure from decreased cardiac output)
• Hepatic failure
• Thyroid disease

Investigations

• Labs: CBC, electrolytes, urinalysis (protein) and microscopy (casts) ◇; urine for quantitative protein, electrolytes, and osmolality ◈
• Imaging: renal US (stone, hydronephrosis, parenchymal disease) ◇; CT ◈

Management

The goal of acute management is to correct life-threatening fluid and electrolyte abnormalities.
• Fluid resuscitation, as needed. If the patient is volume depleted, give boluses (20 ml/kg in children) of crystalloid and monitor response. Caution for overload
• Correct electrolyte abnormalities, especially hyperkalaemia
• Identify contributory processes (e.g. severe sepsis, cardiogenic shock, rhabdomyolysis, hypovolaemic shock, etc.)
• Urine catheter. If difficulty passing per urethra, consider post-renal causes and place suprapubic catheter (📖 p. 824)

Critical documentation

Document initial volume status, fluid intake and output; electrolytes; response to fluid therapy.

Indications for emergency dialysis ◈

• Pulmonary oedema
• Medically uncontrolled severe hyperkalaemia
• Severe metabolic acidosis
• Uraemic pericarditis
• Uraemic encephalopathy
• Dialysable drug overdoses

Disposition

Admit all patients with suspected AKI. In cases of mild pre-renal failure (e.g. diarrhoea with dehydration), if cause resolved and renal function restored, discharge with follow-up. Admit all patients with whose peak creatinine was > twice normal, or whose reduced urine output persisted > 12 hours.

225 Chronic kidney disease

Chronic kidney disease (CKD) is a progressive, irreversible loss of renal function for > 3 months. End-stage renal disease (ESRD) refers to the stage of CKD in which renal replacement therapy (dialysis or transplantation) is required. There are two major types of dialysis. Haemodialysis (HD) involves haemofiltration over a few hours, requires specialised equipment and is usually facility-based. Peritoneal dialysis (PD) involves equilibration over several hours with fluid instilled into peritoneal cavity, is more readily available and is often performed at home.

While CKD itself is chronic, its complications, including hyperkalaemia, volume overload, sepsis or haemorrhage

can be rapidly life threatening.

The first five minutes

- ABC, VS, O2, pulse oximeter, cardiac monitor
- Look for wide complex tachycardia, hyperkalaemia, and pulmonary oedema

History and physical examination

Key historical features

History of renal insufficiency, decreasing urine output, long-standing lower extremity or pulmonary oedema.

Signs and symptoms

- Volume overload: heart failure, hypertension, pericardial tamponade, pulmonary oedema, pleural effusion
- Uraemia: pleuritis, pericarditis, lethargy, somnolence, poor concentration, seizures, uraemic encephalopathy (hiccups, asterixis, myoclonic twitching, AMS), anorexia, nausea, vomiting, uraemic frost (white powder-like skin discoloration), pruritis
- Musculoskeletal: renal osteodystrophy (bone pain, osteoporosis, fractures), spontaneous tendon rupture, arthritis, myopathy
- Immunologic: increased susceptibility to infections
- Haematologic: anaemia (due to lack of EPO), ecchymosis/bleeding tendency (platelet dysfunction), WBC dysfunction leads to infection risk, haemorrhage at fistula site common
- Peritonitis: nausea, vomiting, abdominal pain and a cloudy dialysate effluent

Possible causes and differential diagnosis

Possible causes

Vascular (hypertension, stenosis), medication-induced, glomerular (diabetic, post-infectious inflammatory, HIV), tubulo-interstitial (hypercalcaemia; chronic infections including TB, sickle cell; toxins, including analgesics and other medications), polycystic kidney disease; obstructive (BPH, nephrolithiasis).

Differential diagnosis

CCF, severe hypertension, pulmonary effusion, liver failure, thyroid disease.

Investigations

- Labs: CBC (normocytic normochromic anaemia), electrolytes, raised K), renal ◇, calcium (low), phosphate (high) ◈
- ECG ◇: (signs of hyperkalaemia)

Peritonitis in PD patients

Diagnosis is made by peritoneal fluid analysis: > 100 wbc/mm^3 in peritoneal fluid with > 50% neutrophils, or positive gram stain (only 30–40% positive) ◇. Rule out other causes of abdominal pain (hernias and bowel obstruction common).

Management

The goal of acute management is to normalise volume status to maintain oxygenation and perfusion, control haemorrhage, and to identify and treat electrolyte disturbances and infections.

Hyperkalaemia

(📖 p. 426)

Pulmonary oedema

(📖 p. 104)
• Administer DDAVP or tranexamic acid ◈

> ### Indications for emergency dialysis ◈
> • Pulmonary oedema
> • Medically uncontrolled severe hyperkalaemia
> • Severe metabolic acidosis
> • Uraemic pericarditis
> • Uraemic encephalopathy
> • Dialysable drug overdoses

Specific HD-related complications

• Vascular access-related complications:
  » Bleeding: apply direct pressure, avoid complete occlusion of vessel (can cause thrombosis)
  » Local infection: blood cultures, antibiotics as per local guidelines ◇
• Non vascular access-related complications:
  » Hypotension post-HD: usually resolves spontaneously. May need small volumes of normal saline. Consider other causes of hypotension
  » Disequilibrium syndrome (due to rapid fluid and electrolyte shifts): headache, nausea, vomiting, AMS and seizures – usually resolves within several hours. Rule out other causes (CT to evaluate intracranial haemorrhage (platelets are dysfunctional)) ◈

Specific PD-related complications

Catheter exit site infections – treat with oral antibiotics and cleaning of the site TID with povidone iodine.

Critical documentation

Serial VS, serial K levels, treatments administered and response; peritoneal fluid results (as indicated) in PD patients. Document plan for next dialysis.

Disposition

Admit to nephrology for cases requiring emergency dialysis ◈. If unavailable, refer to appropriate facility after stabilisation. Patients being discharged need appropriate follow-up arrangements.

226 Haematuria

Haematuria is the presence of red blood cells (RBC) in the urine and is a common acute presentation.
• Gross haematuria: urine visibly discoloured by blood (as little as 1 ml in 1 l of urine)
• Microscopic haematuria: > 5 RBC per high power field (HPF). May be glomerular (with casts, suggesting renal origin) and non-glomerular (from renal pelvis, ureter, bladder, or urethra, suggesting urologic disease)

NOTE: Myoglobinuria and bilirubinemia may be reported as haematuria. Myoglobinuria will be dip-positive for blood with no RBC on microscopy (see 📖 Rhabdomyolysis, p. 617).

The first five minutes

• ABC, IV. For shock: IVF, type and cross

- Quickly examine for other serious bleeding
- Ask about anticoagulants

History and physical examination

Key historical features

- Risk factors for urothelial cancer in patients with microscopic haematuria:
  - Smoking
  - Age > 40 years
  - Gross haematuria
  - History of pelvic irradiation
  - History of urological disorder or disease
  - Analgesic abuse
  - History of irritative voiding symptoms
  - Occupational exposure to chemicals or dyes (benzenes or amines)
- Recent skin or other infection (post-infectious glomerulonephritis (p. 383))
- Transient haematuria may result from menstruation or sexual activities in healthy patients
- Vigorous exercise may produce transient haematuria or myogobinuria reported as haematuria
- Family history of renal disease, sickle cell anaemia
- Exposure to schistosomiasis, malaria and TB

Signs and symptoms

Examination should focus on the presence of rash (especially in children), fever, costoverterbral or suprapubic tenderness, hypertension, oedema, palpable abdominal or flank masses. Do a rectal examination in older males to evaluate prostate.

- Glomerular causes of haematuria are usually painless
- Passage of clots in the urine → extraglomerular cause
- Fever, dysuria and frequency → UTI; colicky flank or abdominal pain (without notable tenderness) suggests kidney stones.

Possible causes and differential diagnosis

Most common causes in children are UTI, glomerulonephritis, and congenital abnormalities. Gross haematuria in children may result from HSP or IgA nephropathy.

Most common causes in adults are UTI, bladder cancer, urolithiasis; and BPH in men > 60.

- Urogentital disorders
  - Urinary tract infection
  - Urethritis and prostatitis (p. 612)
  - Urinary calculi
  - Urinary tract malignancy
  - Endometriosis
  - Benign prostatic hypertrophy
  - Anatomical abnormalities
- Renal disorders:
  - Glomerulonephritis
  - Renal artery stenosis
  - Interstitial nephritis
  - Papillary necrosis
  - Ig + nephropathy
  - Alport syndrome
- Systemic disorders:
  - Hyperuricosuria

    » Coagulation abnormalities
    » Diabetes
    » Sickle cell disease
    » Hypercalciuria
- Others:
    » Trauma
    » Menstruation
    » Exercise and fever

Investigations

Consider investigations related to probable differential.
- Labs: CBC, electrolytes, renal, urinalysis (protein, RBC) ◇; PT/PTT ◈
- Imaging: US (renal size, masses, cysts and to rule out obstruction) ◇; intravenous urography (not as sensitive as CT), CT (stones, renal mass) ◈
- Urine microscopy ◇ (dysmorphic RBCs, casts or proteinuria suggest glomerular origin)
- Urine culture ◇ (UTI, schistosomiasis and TB)
- Urethral culture (STI)
- Urine cytology and tumour markers ◈

Management

The goal of acute management is to identify source of blood, rule out life-threatening bleeding, treat infections, and direct to proper follow-up.
- Antibiotics (UTI or infected stone)
- Urine catheter for obstruction (see 🕮 p. 822) ◇ (may require flushing if clots)

Critical documentation

Serial VS, risk factors, amount and progression of haematuria, management and response.

Disposition

Admit unstable, undifferentiated (unclear cause), and febrile patients. Stable patients with benign aetiology may be discharged with follow-up.
Refer to an urologist if:
- Gross haematuria
- Imaging showing urological disease
- Abnormal cytology
- High risk factors despite negative work up
- Cystoscopy needed

Refer to a nephrologist for:
- Acute kidney injury
- Decreased GFR
- Indication for renal biopsy (persistent haematuria, 2+ proteinuria)
- Underlying cause unclear

227 Urolithiasis

Renal calculi are found in the kidneys and ureteric calculi are found in the ureters. The pain caused by the presence of ureteral stones can be excruciating and is termed either renal colic or ureteric colic, depending on the position. The calculi are caused by precipitation of calcium oxalate or calcium phosphate salts in 75% of cases. Strongly consider other diagnoses for first-time presentations in patients over 60.

The first five minutes

Provide analgesia. Exclude life threatening condition (urosepsis, ruptured AAA, etc.).

History and physical examination

Key historical features

• History of prior similar symptoms
• Pain typically radiates from flank to groin or testicles

Signs and symptoms

• Blood tinged urine or frank haematuria
• Severe pain, typically radiating from the flank to the groin, without significant abdominal tenderness to palpation and without testicular tenderness (if present, concern for torsion)
• Costovertebral angle tenderness
• Fever
• Normal pelvic examination in women

Possible causes and differential diagnosis

• Pyelonephritis
• Appendicitis
• Abdominal or gynaecological tumours
• Abdominal aortic aneurysm
• Muscular strain or lower back injuries
• Gallstones
• STIs
• Testicular or ovarian torsion

Investigations

• Labs: CBC, electrolytes, renal, urinalysis (haematuria in 75–85%; pH > 7.5 (consider *Proteus* as cause of calculi), pH < 5 (consider uric acid calculi), or leukocytosis (consider infection)) ◇
• Imaging: AXR (85% of renal calculi visible on XR, though only 60% identified prospectively), US (85% sensitive for hydronephrosis, 60% for stone) ◇; intravenous pyelogram (if US or CT not available, 90% sensitive for hydronephrosis, 72% for stone)), CT (99% sensitive, diagnostic modality of choice) ◈
• Patients with recurrent episode, similar presentation to prior, and no high-risk factors do not require imaging

Management

The goal of acute management is pain relief, maintenance of adequate hydration, and identification of hydronephrosis and infection.
• IV morphine, or other analgesia (NSAID) as needed
• Alpha1-antagonists (tamsulosin or alfuzosin) to facilitate passage ◈
• IVF as needed
• IV antibiotics if suspect infection ◇
• Consult urology for stones > 5 mm and all stones with signs of infection ◈

Critical documentation

Document signs of infection, analgesia administered and response.

Disposition

• Admit if signs of infection, severe pain (not responding to opiates), persistent vomiting, single or transplanted

kidney, diabetic, or calculus > 10 mm
• Discharge all other patients with outpatient follow-up

228 Urinary tract infection and pyelonephritis

Simple urinary tract infections (UTIs) are rarely life threatening. However, they can lead to serious conditions like pyelonephritis and urosepsis. Both are most commonly caused by *Escheria coli* (70–95%), with the remainder caused by *Proteus*, *Klebsiella*, *Staphylococcus saprophyticus* and enterococcus. (See 🕮 Urinary tract infections in children, p. 606.)

The first five minutes

• ABC, VS
• Look for urosepsis; IVF as needed

History and physical examination

Key historical features

• Any history of urologic infection or disease
• Fever
• Recent catheterisation or antibiotics

Signs and symptoms

UTI

• Burning pain on urination
• Frequent, small volume urination
• ± Haematuria
• No or mild fever, no flank pain, no systemic signs/symptoms
• Exam should be normal ± mild suprapubic tenderness
• No costovertebral angle tenderness or vaginal discharge

Pyelonephritis

• Signs of UTI plus fever > 38.5°C, flank pain or abdominal pain
• Costovertebral angle tenderness is insensitive and non-specific

Possible causes and differential diagnosis

• HSV infection or other STI, especially if recurrent. In men, prostatitis or anatomic abnormality should be considered
• Schistosomiasis – consider with micro/macroscopic haematuria, even with concurrent UTI
• Urinary TB

Investigations

Burning pain on urination is sufficient for UTI diagnosis if dipstick is not available.
• Labs: electrolytes, renal (if pyelonephritis), urinalysis (nitrites (indicates enterobacteria) and/or leukocytes indicate UTI) ◇
• Urine microscopy and culture if dipstick is positive (especially in children and pregnant women) ◈

Management

The goal of management is treatment of the infection to prevent progression to pyelonephritis or urosepsis.

- Analgesia and/or an anti-pyretic as needed
- Resistance to antimicrobial agents is VERY common, and treatment will vary based on local susceptibilities. See 🕮 Antibiotic guidelines p. 968 for general recommendations
- For pyelonephritis, IVF for hypotension/dehydration ◇ and paracetamol for fever as needed

Critical documentation

Document urine dipstick and other results, abnormalities on examination, management.

Disposition

- Discharge well patients with UTI; discharge and follow-up patients with pyelonephritis if no signs of serious illness. ALL patients with pyelonephritis should receive antibiotics prior to discharge as gram-negative sepsis can be evolve rapidly
- Admit all unwell patients, patients with pyelonephritis and vomiting or hypotension, and all patients where there is concern for medication availability or compliance

229 Urinary tract infections in children

UTI is common in children, and vesico-ureteral reflux is a common risk factor. In neonates, UTI may be due to haematogenous spread and is often associated with sepsis. Under one month, UTI is more common in boys, but is seen more frequently in girls thereafter. *E.coli* is the main causative organism.

The first five minutes

- ABC, VS
- Look for urosepsis; IVF as needed

History and physical examination

Key historical features

- Other causes of fever (i.e. upper respiratory infection, otits media) as may be concurrent with UTI
- History of similar symptoms or prior diagnosis of UTI
- Any recent antibiotics

Signs and symptoms

Varies by age group:
- Neonates: fever, lethargy, irritability, and poor feeding
- Older children: restlessness, fever, mild abdominal/suprapubic tenderness, dysuria, frequent urination and nausea
- Look for concurrent pyelonephritis or sepsis

Possible causes and differential diagnosis

Possible causes

- Bacteria, fungus, pinworms, schistosomiasis
- Vesiculoureteral reflux
- Labial adhesions, renal stones
- Sexual intercourse in adolescents

Differential diagnosis

- Appendicitis
- Pyelonephritis

- Gastroenteritis
- Vulvovaginitis (irritation from new soaps or foreign body)
- Trauma: sexual abuse, masturbation, straddle injury
- Wilm's tumour
- Pinworms
- Ureteric calculi

Investigations

Use urethral catheterisation in patients < 2 years to obtain samples (5Fr feeding tube can be used). ◇ Bag collection is not reliable.

- Labs: urinalysis (leucocytes, nitrites or blood) ◇
- Imaging: renal and bladder US (infants – rule out anatomic abnormalities) ◇
- Urine microscopy, culture and sensitivity ◇

Management

The goal of acute management is treatment of the infection to prevent progression to pyelonephritis or urosepsis. Early treatment should be given to prevent renal scarring.

- Analgesia and/or an anti-pyretic
- See 🕮 Antibiotic guidelines p. 968 for recommended therapy
- Refer to urology for recurrent UTIs or anatomic abnormalities ◈

Critical documentation

Document the age, weight and VS; signs of complicated UTI (very young age, fever, hypotension, tachycardia, flank pain) or any signs of sepsis; management administered.

Disposition

Admit:

- All children < 1 year
- Suspected pyelonephritis
- Inability to tolerate oral meds or fluids
- Known sickle cell disease
- Diabetics
- Patients with fever, tachycardia or hypotension

Discharge other children with UTI with antibiotics as above; first dose antibiotics prior to discharge strongly recommended.

230 Penile problems

Patients with any penile trauma or priapism should be evaluated urgently. See also 🕮 Urogenital trauma p. 752 and Priapism p. 610).

The first five minutes

- Identify trauma and surgical emergencies
- Remove foreign bodies around shaft or in urethra immediately as progressive oedema may compromise perfusion
- Analgesia

History and physical examination

Key historical features

Onset of pain, lesions, trauma, sexual activity, penile discharge, dysuria, haematuria, systemic symptoms.

Signs and symptoms

• Inspection and palpation of the abdomen, inguinal and groin areas, penis, scrotum/testis, and prostate
• Uncircumcised boys and men may develop phimosis and paraphimosis
• Penis:
  » Examine for ecchymosis, haematoma, lacerations, avulsions, bleeding or evidence of urethral trauma, degloving injuries, lesions, swelling, and deformity of the shaft or glans
  » Foreign bodies in urethra or around shaft
  » If uncircumcised retract foreskin and expose glans
  » Discoloured skin, foul odour, crepitus in Fournier's gangrene
  » Gross inspection of urine for blood and pus

Possible causes and differential diagnosis

Paraphimosis, penile trauma, priapism, Fournier's gangrene, foreign body, phimosis, balanitis, posthitis, penile fracture, urethritis, STI (syphilis, *Neisseria gonorrheae, Chlamydia trachomotis*, chancroid).

Investigations

• Labs: urinalysis ◇; others per clinical presentation
• Imaging: retrograde urethrogram (RUG) (if concern for urethral trauma) ◈

Management

The goal of acute management is identification and rapid correction of compromised perfusion, and treatment of organ- or life-threatening infection.

Provide analgesia; use ice for oedema.

Penile trauma

See also 📖 Urogenital trauma, p. 752, penile fracture, urethral laceration, haematoma, vascular injury.
• Control haemorrhage and oedema with pressure and ice
• If blood at urethral meatus evaluate for urethral injury with RUG (do not place Foley) ◈
• Surgical evaluation and early intervention for acute penile trauma

Paraphimosis

Foreskin becomes retracted, oedematous, and swollen in uncircumcised males constricting the glans and leading to ischaemia and necrosis.
• Control pain (oral, topical, or block) and reduce penile oedema with compressive dressing, manual pressure, or ice; or sprinkle granulated sugar on prepuce and glans (for osmotic reduction of oedema)
• Manual reduction
• Puncture technique – multiple punctures with needle to drain foreskin
• Emergency dorsal slit to reduce constriction of glans

Priapism

📖 p. 610.

Phimosis

The prepuce cannot be retracted proximally over the glans. (Severe phimosis may result in urinary obstruction and preputial stone formation.) If obstruction or stone formation, may require dorsal slit excision.

Balanitis

Inflammation of the glans penis with redness and oedema. Control blood glucose in diabetics. Co-infection common: treat with antibiotics and antifungal (topical OK).

Urethritis

- Treat for *Neisseria gonorrhoeae* and *Chlamydia trachomatis* based on local sensitivities (sexually active patients)
- Give antibiotics for common urinary pathogens in older patients

Fournier's gangrene

Necrotising infection of penis, testicular sac, and perineum. See 🕮 Testicular pain, p. 614.
- Broad spectrum intravenous antibiotics with anaerobic and aerobic coverage ◇
- Surgical debridement of necrotic tissue ◇

Critical documentation

Time seen by provider, duration of acute penile pain or erection, time surgical team informed and arrived, time and name of antibiotics given.

Disposition

Admit Fournier's gangrene to surgery. Consider admission of other surgical emergencies (dorsal slit, etc.).

231 Priapism

Priapism is a medical emergency, and is defined as 'persisting, painful and abnormal tumescence'. It can occur without sexual stimulation and does not subside after ejaculation. It can be low-flow (ischaemic, more common and more dangerous); or high flow (non-ischaemic). The risk of permanent damage and impotence increases with time untreated (complications increase significantly after four hours). Priapism is a well-documented complication of sickle cell disease.

The first five minutes

ABC, VS, IV, IVF (if sickle cell disease), analgesia.

History and physical examination

Key historical features

- High flow: direct injury to the penis or genital area; usually not painful
- Low flow priapism: painful

Signs and symptoms

Persistent, painful erection. If not painful, look for signs of spinal trauma.

Aspiration of blood from the corpora cavernosa will help distinguish low-flow priapism from high-flow priapism. If the aspirated blood is bright red and is well oxygenated, then it is suggestive of high-flow priapism. Dark blood is suggestive of low-flow priapism.

Possible causes and differential diagnosis

High flow priapism is usually due to a direct injury to the penis or genital area. Causes of low-flow include:
- Spinal trauma
- Sickle cell disease

- Leukaemia
- Intracavernosal injection of agents
- Medications (e.g. anti-depressants, anti-coagulants, anti-hypertensives, phenothiazines, sildenafil, etc.)
- Drugs of abuse (cocaine and alcohol)

Investigations

A clinical diagnosis.

Management

The goal of acute management is to relieve priapism and prevent ischaemic damage to the penis.
Establish if high-flow or low-flow.

- High flow: urgent referral to urology (if available); if no urological service, careful monitoring for any evidence of ischaemia
- Low flow: urgent referral to urology, plus phenylephrine/adrenaline as below or aspiration and irrigation ◇

Treatment of low-flow (ischaemic) priapism

- Step 1: IV access
- Step 2: penile dorsal nerve block for anaesthesia
- Step 3: drain ~5 ml to decompress corpora
- Step 4: intra-corporeal phenylephrine (preferred if available) or adrenaline injection:

  In adults, phenylephrine should be diluted with normal saline to provide a final concentration of 100–500 mcg per ml. Serial doses of 1 ml of dilute solution can be given Q5 min up to one hour.

  If phenylephrine not available: 0.1 mg adrenaline (0.1 ml 1:1 000) in 2 ml normal saline; inject into the corpus at either the 10 o'clock or the 2 o'clock position at the base of the penis. Wait for 30 minutes for a response. If unsuccessful, follow step 5.
- Step 5: aspirate corpora cavernosum: using sterile technique, insert a small (22 g or smaller) needle into one of the copora cavernosa. Aspirate 20–30 ml of blood. If the penis remains erect, follow step 6.
- Step 6: irrigate with saline or solution of adrenaline (add 1 mg of adrenaline to 1 l of normal saline and irrigate with 20–30 ml of this solution at a time).

Sickle cell disease

O_2, IVF, IV morphine ◇; exchange transfusion if available ◈.

Critical documentation

Document duration of symptoms, cause, low-flow or high-flow, management and response.

Disposition

Admit all patients with persistent or recurrent symptoms. Refer all for urgent urological assessment.

232 Prostatitis

Prostatitis is an inflammation of the prostate gland; it comprises multiple syndromes that affect the prostate, including acute and chronic bacterial prostatitis, chronic abacterial prostatitis, and asymptomatic inflammatory prostatitis.

The first five minutes

Resuscitate as needed (acute prostatitis with signs of sepsis or acute renal failure). Avoid repeat exams/prostatic

massage as it may induce or aggravate sepsis.

History and physical examination

Key historical features

- Pain of perineum, lower abdomen, testicles, and/or penis and pain with urination/ejaculation
- Frequent urination (especially at night) or urinary 'urgency'

Specific clues based on condition:

- Acute bacterial prostatitis is associated with fever and chills
- Chronic bacterial prostatitis is associated with recurrent UTI and blood in semen
- Chronic abacterial prostatitis presents with similar symptoms in the absence of positive urine or semen cultures
- By definition, patients with asymptomatic inflammatory prostatitis lack symptoms

Signs and symptoms (non-specific)

- Enlarged or tender lymph nodes in groin
- Lower abdominal tenderness
- Enlarged bladder (due to urinary retention)
- Prostate may be tender, nodular, boggy, hot or normal on digital rectal examination

Possible causes and differential diagnosis

- Urinary tract infection
- Urethritis
- Urinary incontinence
- Benign prostatic hyperplasia
- Prostate cancer

Investigations

Presumptive diagnosis is clinical. Investigations may guide management.

- Labs: CBC, electrolytes, renal, urinalysis
- Imaging: US (detect volume of retained urine; suspected prostatic abscess) ◇; CT (prostatic abscess or neoplasm) ◈
- Urine microscopy and culture ◇

Management

The goal of acute management is relief of symptoms, eradication of infection, and modification of risk factors (e.g. unprotected sex) if present.

Acute bacterial prostatitis

- Supportive measures such as antipyretics, analgesics, hydration and stool softeners may benefit. Antibiotic selection will depend on local resistance patterns in sexually active patients. Use STI regimen (📖 p. 356)
- Oral antibiotics: 14–28 days of treatment with oral antibiotics, including fluoroquinolones or trimethoprim-sulfamethoxazole
- Parenteral antibiotics: broad-spectrum penicillins, cephalosporins with or without aminoglycosides, or fluoroquinolones ◇

Chronic bacterial prostatitis

Commonly requires longer treatment course with antibiotics and relapse is common.

- Fluoroquinolones or trimethoprim-sulfamethoxazole are preferred and prescribed for minimum four weeks

- Refractory cases or difficult to treat organisms may require treatment up to 12 weeks
- Analgesics and stool softeners
- Alpha blockers (terazosin or doxazosin), may help with symptom relief ◇
- If available, treatment should be guided by the results of urine culture ◇

Chronic abacterial prostatitis

- Management varies, usually give a trial of antibiotics and alpha-blockers with re-evaluation in one and two months
- Analgesia

Asymptomatic inflammatory prostatitis

- Trial of antibiotics and/or anti-inflammatories are often prescribed, but have limited efficacy

Critical documentation

Document digital rectal examination findings, treatment provided.

Disposition

Admit patients who appear acutely ill, have evidence of sepsis, or have urinary retention. Manage others as outpatients, with urology follow-up where available.

233 Testicular pain

The commonest aetiologies of acute testicular pain are testicular torsion, torsion of a testicular appendage, and epididymitis. Testicular pain is commonly referred to the abdomen or inguinal area. Testicular salvage rate with torsion is over 90% with surgical treatment within six hours of pain onset, but decreases rapidly after this interval.

The first five minutes

- ABC, VS, O_2, IV, analgesia
- If torsion suspected, immediate surgical consultation – do not delay consult for workup

History and physical examination

Key historical features

- Timing of onset of pain
- Trauma, sexual activity, penile discharge, dysuria, haematuria
- Vaccination status (mumps)
- Testicular pain with fever, nausea or vomiting is of particular concern in torsion

Signs and symptoms

Torsion

- Sudden onset of scrotal, inguinal, or abdominal pain due to ischaemia from twisting of spermatic cord
- Abdominal pain complaints may predominate
- Small peak in neonates and larger peak in post-pubertal boys (≥ 12 yrs), but can happen at any age

Torsion of the appendage of testis

- Sudden or gradual onset of testicular pain
- More common in pre-pubertal age (3–12 year old)

Epididymitis

• Gradual onset of pain over days
• All ages, but more common in post-pubertal boys and adults

Fournier's gangrene

• Life threatening necrotising infection of genitals and perineum
• Risk factors include immunocompromise, diabetic, alcoholic and elderly patients
• Insidious onset of perineal pain, nausea/vomiting, then rapid progression

Signs and symptoms

• Inspect and palpate abdomen, inguinal areas, penis, and scrotum/testis
• Examine for inguinal hernias (reducible, painful?) and boggy or tender prostate
• Inspect the urine for frank blood or pus

Testis

• Testicular swelling, tenderness, masses, or size discrepancy
• 'Blue dot sign' (a tender blue or black spot beneath the scrotum skin) is pathognomonic for torsion of a testicular appendage
• Tender, high riding, and horizontal testicle associated with testicular torsion
• Tender, swollen epididymis – epididymitis (orchitis – tender, swollen testicle)
• Torsion in neonates – irritable with a dusky, swollen testicle (does not transilluminate)

Scrotum

• Pain, swelling, crepitance, gangrene (Fournier's)
• Note scrotal lesions, discoloration, ecchymosis, erythema, induration (cellulitis, STI)

> Absent or decreased cremasteric reflex is most sensitive physical exam finding for testicular torsion; however, it is nonspecific and its absence does not rule out torsion.

Possible causes and differential diagnosis

Torsion of testicular appendage, epididymitis, Fournier's gangrene, hernia, testicular trauma, orchitis, varicocele, hydrocele, neoplasm, HSP, ureteric stone, AAA (with compromise of testicular perfusion).

Investigations

Mainly clinical diagnosis; pre-operative blood tests as needed.
• Labs: urinalysis (pyuria with or without bacteria (suggestive of epididymitis)) ◇, pre-operative blood typing
• Imaging: Doppler US of testes for blood flow ◇
• Bedside US in affected versus normal testes to compare flow

Management

The goal of acute management is to identify life- or testicle-threatening conditions, restore flow, and treat infection. Provide analgesia, keep NPO, and consider acute surgical referral.

Testicular torsion

• Emergency surgical exploration
• If surgery not immediately available, try manual detorsion (rotate affected testicle medial to lateral)

Torsion of testicular appendage

• Self-limiting. Control pain, reduce activities, NSAIDs

Epididymitis/epididymo-orchitis

• Treat for *Neisseria gonorrhoeae* and *Chlamydia trachomatis* based on local sensitivities in sexually active adults
• Cover for common urinary pathogens in older patients
• NSAIDs
• If signs of systemic infection – IV antibiotics ◇

Testicular trauma

Torsion, penetrating injury, testicular rupture, or dislocation
• Analgesia
• Acute referral for surgical evaluation

Fournier's gangrene

• Resuscitation
• Broad spectrum IV antibiotics with anaerobic and aerobic coverage (ceftrioxone or cefotaxime) ◇
• Emergency surgical debridement of necrotic tissue

Critical documentation

Document time of pain onset, investigation results, management.

Disposition

• Admit unwell patients, or those needing parenteral analgesia
• Admit to surgery Fourniers gangrene, testicular torsion, and trauma
• Discharge well patients with epidymoorchitis or appendage torsion

234 Rhabdomyolysis

Rhabdomyolysis refers to the breakdown of skeletal muscle fibres, which releases myoglobin into the blood stream. If present in sufficient quantities, kidney damage can result. Aggressive early treatment can help save renal function. See also 🕮 Crush syndrome p. 774.

The first five minutes

• ABC, VS, O_2, IVF, cardiac monitor
• ECG (look for hyperkalaemic changes)

History and physical examination

Key historical features

Patients usually present with muscle weakness and pain. They may complain of tea-coloured urine or 'blood' in urine. Ask about trauma, seizures, medications, recent illness.

Signs and symptoms

Muscle tenderness on palpation may be present. Examine all muscle groups for possible compartment syndrome.

Possible causes and differential diagnosis

Possible causes

- Crush syndrome (📖 p. 774)
- Compartment syndrome
- Extreme physical exertion
- Prolonged seizures or status epilepticus
- Hyperthermia
- Medications (e.g. statins, antipsychotics, SSRIs)
- Viral infections
- Polymyositis, dermatomyositis
- Prolonged immobility

Differential diagnosis

Exclude haematuria and bilirubinuria.

Investigations

- Labs: electrolytes, renal, urinalysis (bilirubin, blood) ◇; CK ◈; urine myoglobin (if available) ◈
- Urine microscopy to evaluate for RBCs (dipstick will be positive for blood with myoglobinuria, but no RBCs on micro) ◇
- ECG (hyperkalaemia) ◇

Management

The goal of acute management is to protect the kidneys from damage, and to recognise and treat compartment syndrome.

- Administer large volumes of fluid. Normal saline is preferred; aim for urine output of ~200 ml/hr
- Monitor for signs of compartment syndrome
- If severe rhabdomyolysis with climbing CK despite IVF, consider sodium bicarbonate (50 mEq in half-normal saline) to target urine pH > 6.5. Watch carefully for hypocalcaemia. Stop for serum pH > 7.50 or serum bicarbonate < 30 meq/L. Unclear benefit
- Monitor K levels; manage electrolyte imbalances as necessary

Critical documentation

Strictly record fluid intake and urine output; document serial VS, electrolytes, treatment administered and response.

Disposition

Admit all patients.

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Part

4

P. Respiratory system

235 Approach to the adult with cough

Cough is one of the most common acute presentations; it usually results from irritation (from mass, effusion) or infection. Cough is a symptom, not a disease, and it is important to search for the cause. (📖 Difficulty in breathing p. 54)

The first five minutes

- ABC, VS, O_2 as needed to keep sat > 94% (📖Haemoptysis, p. 632

History and physical examination

Key historical features

- Onset: sudden or gradual
- Description: productive vs. nonproductive, bloody
- Duration of cough: acute (< 3 weeks), subacute (3–8 weeks), chronic (> 3 weeks)
- Associated symptoms: dyspnoea, chest pain, leg swelling, lethargy, constitutional symptoms (weight loss, night sweats)
- Signs of infection: fever, chills, rhinorrhea
- Other: asthma, CCF, HTN, recent surgery, malignancy, PE, PTX, lung disease
- TB exposure, HIV status (CD4 count)
- Medications: ACE-inhibitors, immune-suppressors
- Social history: smoking, travel

Signs and symptoms

- Palpitation: crepitus, rib fracture
- Inspection: chest expansion, chest wall motion, retractions, barrel chest, chest wall abnormalities
- Percussion: dullness, resonance; note differences between sides
- Auscultation: bronchial sounds, wheezing, rhonchi, crepitations
- Cardiac exam: gallops, rubs, murmurs, peripheral oedema

- Extremities: clubbing, splinter haemorrhages, rash
- Signs of chronic disease: wasting, cachexia, skin rashes, pallor, hair changes

Differential diagnosis

Duration of symptoms can help identify aetiology of disease.

Acute (< 3 weeks)	Subacute (3–8 weeks)	Chronic (> 8 weeks)
Infection (lower or upper airways)	TB	TB
Congestive cardiac failure	Lung abscess	Lung abscess
Pulmonary embolus	Chronic bronchitis	Malignancy (mediastinal or pulmonary)
Foreign body aspiration	Post-viral bronchospasm	ACE-inhibitors
Asthma (nocturnal cough)	Foreign body	Chronic bronchitis
COPD	Asthma	Bronchiectasis
Bronchiectasis	*Bordetella pertussis*	Interstitial lung disease
Acute bronchitis		Asthma
Influenza		Gastro-oesophageal reflux disease
Sinusitis		
Bordetella pertussis		
Allergic rhinitis		
Upper airway cough syndrome (due to post-nasal drip)		

Investigations

- Labs: CBC (leucocytosis, anaemia), ABG (determine CO_2 level and acid-base status) ◇; D-dimer (PE), cardiac markers ◈
- ECG ◇, (cardiac aetiology)
- Imaging: CXR ◇ (pneumonia, CCF, TB, mass, foreign body); CT chest ◈ (PE, malignancy, interstitial disease)
- Peak flow (serial measures for response to treatment)
- Sputum: acid-fast bacillus (TB)

Management

The goal of acute management is symptom relief and identification of dangerous aetiologies:

- Pneumonia – antibiotics (🕮 p. 332)
- CCF – diuresis (🕮 p. 104)
- PE – anticoagulation (🕮 p. 118)
- URI – symptomatic treatment, antihistamine, decongestant (pseudoephedrine)
- Acute bronchitis – consider bronchodilators (beta-agonists); no role for antibiotics unless underlying COPD
- *Bordetella pertussis* – macrolide antibiotic (implicated in up to 30% of adults with chronic cough as vaccine immunity has been found to wane faster than expected; pertussis vaccine is now provided in combination with tetanus)
- Influenza – consider antivirals (oseltamivir)
- Lung abscess – antibiotics (prolonged course; including anaerobic coverage), drainage (refer to surgery)
- TB – respiratory isolation; setting-specific management protocols (🕮 p. 337)
- Asthma and post-viral bronchospasm – beta2-agonists and steroids (🕮 p. 626)
- Foreign body – ENT or gastroenterology consult
- Gastro-oesophageal reflux disease – H2-blockers, lifestyle modification

236 Approach to the child with cough

Cough has multiple causes, and usually presents with associated findings. Acute infections are the most common cause. Hypoxia in room air (O_2 sat < 92%) indicates severe respiratory illness that needs urgent attention.

Cough is a symptom, not a disease and it is always important to search for the cause. (🕮 RAP Difficulty in breathing p. 52)

The first five minutes

• ABC, VS, O_2 sat, O_2 as needed

History and physical examination

Key historical features

- Duration of cough and pattern of onset (sudden vs gradual)
- History of underlying medical illness, medications, malnutrition
- Recent vomiting, diarrhoea, difficulty feeding, lethargy or seizures
- HIV status or previous HIV testing in child and mother
- Exposure to TB contact

Signs and symptoms

- Presence of probable infection (e.g. fever, runny nose)
- RR, effort of breathing, distress or exhaustion (impending respiratory failure)
- Tachypnoea, tachycardia, poor perfusion; hypoxia, fever; pallor
- Presence of ‘noisy breathing’ with expiration (wheeze), or inspiration (stridor – from pharynx, sounds like snoring – or stertor – from airway, sounds like croaking)
- Evidence of hyperinflation or air trapping on percussion
- Symmetry of breath sounds (asymmetrical more likely in pneumonia, malignancy, foreign body and congenital pulmonary abnormalities)
- Abnormal auscultation (wheezing, bronchial breathing and/or crackles)
- Signs of HIV infection, e.g. LAN, oral thrush, skin rashes
- Signs of chronic lung disease, e.g. clubbing, chest deformity, Harrison’s sulcus
- Abnormal heart sounds, enlarged heart (consider pulmonary oedema)

Investigations

Cough is a common symptom and should not be over-investigated when isolated and short-lived in well-appearing, low risk children.

- Labs: ABG (only if CO_2 level and acid-base status needed. Oxygenation is measured with pulse oximeter), HIV (all children with severe symptoms in whom HIV status is unknown) ◇; further testing should be guided by the clinical context
- Imaging: CXR (all children with cough and associated severe disease, particularly in high TB/HIV prevalence settings) ◇

Clinical approach and management

Clinical findings	Likely diagnosis	Management
Fever ; normal RR; normal chest examination, Upper respiratory tract signs, e.g. erythema, exudate, nasal congestion	Upper respiratory tract infection	Symptomatic treatment (📖 Otitis media, p. 182, 📖 Pharyngitis, p. 178, 📖 Tonsillitis, p. 178)
Fever, fast breathing, recessions ± hypoxia	Pneumonia	Antibiotics (📖 Pneumonia in children, p. 334)
Fever, very fast breathing with normal auscultation, cyanosis/severe hypoxia; diffuse infiltrates on CXR; HIV < 1 year old	Consider PCP	Co-trimoxazole plus broad spectrum antibiotics; oral corticosteroids (📖 Pneumonia in children, p. 334 and Fever in immunocompromise, p. 310)
Fever, fast breathing and pleural effusion	Parapneumonic effusion/empyema; TB pleural effusion	Chest tube if empyema, large effusion or very ill. Diagnostic aspirate if well or small effusion. (📖 Effusion, p. 634)
Pneumonia with pyopneumothorax or pneumatoceles	*Staphylococuus aureus* infection	Include cloxacillin (📖 Pneumonia in children, p. 334)
Lobar pneumonia; intrathoracic lymph node enlargement ± large airway compression/displacement ± atelectasis	Pulmonary TB	📖 Tuberculosis, p. 337
Fast breathing, wheezing; hyperinflation of	Bronchiolitis	Supportive treatment and close observation. Short

both lungs ± crackles ± fever; < 2 years		acting bronchodilators or 3–5 % hypertonic saline. Steroids no proven benefit
Fast breathing , wheezing, hyperinflation ± atopy in child ± fever; > 2 years	Asthma Viral triggered wheeze	Asthma, p. 626
Wheezing or cough after choking	Foreign body aspiration	Foreign body, p. 256
Fever, stridor ± barking cough	Croup	Croup, p. 174
Recurrent or chronic cough without danger signs , fever or signs of chronic lung disease	Chronic rhinosinusistis, asthma, aspiration syndrome or gastro-oesophageal reflux	Refer to paediatrics for further investigation and treatment
Cardiomegaly, cardiac murmur, gallop rhythm, hepatomegaly ± oedema	Cardiac failure or cardiac disease	Treat acute cardiac symptoms. Refer paediatrics.

237 Asthma

Asthma is a chronic disease with airway inflammation leading to bronchial smooth muscle hyper-responsiveness and mucosal abnormalities, characterised by acute attacks of varying severity. Patients who have severe attacks while on their regular therapy are at higher risk of poor outcomes, especially if on oral steroids.

The first five minutes

• ABC, VS, O_2, pulse oximetry, cardiac monitor

History and physical examination

Key historical features

• Onset: acute or gradual
• Triggers: URI, allergic exposures, taking medications regularly?
• Past occurrences: need for steroids, emergency visits, hospitalisation, intubation (all are markers for severe disease)
• Most recent steroids?

Signs and symptoms

• Symptoms: dyspnoea, cough, wheezing
• Tachycardia, tachypnoea, hypoxia (late and ominous sign)
• Use of accessory muscles
• Wheezing, prolonged expiration
• Decreased air movement on auscultation (note that in severe cases wheezing may be inaudible until treatment partially opens airways)
• Cough (may be the only sign of bronchospasm)
• Inability to complete sentences without inhalation
• Signs of respiratory failure: AMS, inability to speak, cyanosis
• Unilateral decreased breath sounds: consider associated PTX

Differential diagnosis

CCF, COPD, pneumonia, bronchiolitis, upper airway obstruction, foreign body, PTX, anaphylaxis and PE.

Investigations

• Peak expiratory flow rate:
  » Coach patient for three attempts at best effort
  » 50% below patient's normal value indicates severe attack
  » Monitor changes over time
• Labs: ABG ◇ (limited role; mild/moderate attack – decreased pCO_2; severe attack and respiratory failure –

increasing pCO_2)
• Imaging: CXR ◇ (limited value identify other conditions (e.g. pneumonia, PTX)) ◇

Management

The goal of acute management is to reverse bronchospasm and improve ventilation. Provide O_2 to all patients.

Mild-moderate attack

• Beta-agonists: reverse airway obstruction:
  » Metred dose inhalers (MDI) and nebulisation are equally effective, if patient can comply, spacers improve efficiency of MDI treatment
  » Salbutamol MDI: usual dosing 2 puffs every 2–6 hours as needed
• Steroids: oral prednisolone 1–2 mg/kg daily to max of 60 mg daily for 3–5 days

Severe attack

• Beta-agonists: salbutamol:
  » MDI: 2 puffs at least every 15 min (10 puffs as replacement for nebs)
  » Nebulisation: 2.5 mg every 15 minutes or continuous
• Steroids: require 6 hours to maximum effect
  » Use IV if patient unable to tolerate oral medications
• Anticholinergics: nebulised ipratropium 500 mcg every 15 min for 3 doses – then Q6h
• Magnesium sulphate (conflicting evidence for efficacy): 2 mg IV (50 mg/kg in children) over 20 minutes (risk of hypotension with more rapid infusion)
• For worsening respiratory failure, consider:
  » Adrenaline: 0.3 mg (0.15 mg in children under 30 kg) IM 1:1 000
  » Non-invasive ventilation: CPAP or BiPAP, if available ◈ or ◇
  » Intubation ◇

Ineffective therapies

• Methylxanthines, inhaled glucocorticoids (ineffective for acute attack), antibiotics

Critical documentation

Document past similar events, current therapy, recent steroid treatment, serial VS, response to therapy, initial pulmonary exam and serial peak flow. Continued reassessment and repeated documentation.

Disposition

• Mild: with inhaled beta-agonists and oral steroids
• Moderate: observe or admit
• Severe: admit to ICU ◈
• Admit all patients who were already taking oral steroids at the time of the attack

238 Chronic obstructive pulmonary disease

Chronic obstructive pulmonary disease (COPD) is a smoking-related chronic and progressive disease characterised by airflow limitation and inflammation that is not fully reversible. COPD is increasing in prevalence and is one of the most common causes of death worldwide. Pathologic features are attributed to both chronic bronchitis (inflammatory) and emphysema leading to lung tissue destruction.

The first five minutes

• ABC, VS, O_2, pulse oximtery, cardiac monitor

History and physical examination

Key historical features

- Triggers: smoking, respiratory infections, allergens
- Chronic symptoms: weight loss, decreased exercise tolerance

Signs and symptoms

- Current symptoms: dyspnoea, wheezing, cough, sputum, haemoptysis, fever
- Tachycardia, tachypnoea, hypoxia, respiratory distress
- Wheezing or rhonchi, crepitations, prolonged expiration
- Decreased air movement on auscultation
- Hyper-resonance
- Signs of respiratory failure: somnolence, altered mental status, inability to speak, cyanosis, exhaustion
- Cor-pulmonale
- Look for evidence of PE and PTX

Differential diagnosis

Asthma, pneumonia, CCF, PE and PTX.

Investigations

- Labs: limited utility. ABG (clinical judgment more valuable than specific values; most useful to evaluate degree of CO_2 retention) ◇
- ECG ◇: (usually sinus tachycardia; evaluate for ischaemia, arrhythmia, right heart strain, cor pulmonale)
- Imaging: CXR (determine comorbid conditions (pneumonia, PTX)) ◇

Management

The goal of acute management is to reverse bronchospasm and improve ventilation.

- Mild attacks: treat with O_2, inhaled beta-agonists, ipratropium and oral steroids (prednisolone: start 40–60 mg daily with 7–10 day taper)
- For moderate to severe attacks, use IV steroids, and add antibiotics (doxycycline 100 mg BD 10–14 days). For signs of respiratory failure, use NIPPV (CPAP or BiPAP) ◈ early, and intubation and ventilation as needed ◈

Note: spacer devices improve the efficiency of MDIs.

Critical documentation

Serial VS including pulse oximetry and exams; diagnostic study results and plan. Document all interventions and response to therapy, including peak flow relative to baseline.

Disposition

- Mild: discharge with close follow-up
- Moderate: observe or admit
- Severe: admit to a monitored or ICU bed ◈
- Patients without substantial clinical response within a few hours should be admitted
- All patients already on oral steroids at the time of attack should be admitted

239 Dyspnoea

Dyspnoea is the sensation of shortness of breath or breathlessness. It may result from a range of pulmonary, cardiac and CNS pathology that can be non-urgent to life threatening. It is a symptom, not a disease; institute general management as outlined below and evaluate for reversible causes. (📖 Difficulty in breathing, p. 54.)

The first five minutes

ABC, VS, O_2, pulse oximetry, cardiac monitor

History and physical examination

Key historical features

- Onset and timing: acute/chronic/recurrent
- Course: transient (pneumonia); recurrent episodic (asthma, COPD); progressive (COPD, pneumoconiosis, interstitial lung disease, mesothelioma and other cancers)
- Provocation/palliation
- Associated: fever, cough, haemoptysis, chest pain, leg swelling
- Prior symptoms and risk factors: smoking, trauma, constitutional symptoms, occupational history

Signs and symptoms

- Pulmonary:
  - » Inspect: accessory muscle use; trauma; tracheal shift; symmetric chest wall rise
  - » Palpate: crepitus; rib fracture or flail chest
  - » Chest expansion, fremitus
  - » Auscultate: air movement; stridor, wheezing, rales or rhonchi
- Cardiac: muffled heart sounds; murmurs, rubs or gallops; displaced apex beat; elevated JVP
- Neurological: AMS, weakness, sensory deficit, asterixis
- Extremities: peripheral oedema; evidence of DVT
- Skin: diaphoresis or cyanosis

Identify signs of impending respiratory failure

- AMS or ALoC
- Inability to speak more than 2–3 word sentences
- Cyanosis
- Severely increased DIB
- Fatigue compromising respiratory effort

Differential diagnosis

Table 239.1 Differential diagnosis of dyspnoea

Organ system	**Acute dyspnoea**	**Chronic dyspnoea**
HEENT	Foreign body, anaphylaxis, epiglottitis, retropharyngeal abscess, Ludwig's angina, angio-oedema, trauma	Malignancy
Pulmonary	Asthma, COPD (exacerbation), PE, pneumonia, influenza, bronchitis, bronchiolitis, TB, ARDS, PTX, non-cardiogenic pulmonary oedema	COPD (progressive), pleural effusion, cystic fibrosis, ILD, pneumoconiosis, asbestosis, silicosis, bronchiectasis, malignancy
Cardiac	CCF (exacerbation), acute LV failure (HPT, ACS), MI, tamponade, pericarditis, dysrhythmia, acute valve dysfunction	CCF (progressive)
Neurological	Neuropathies, phrenic nerve injury, other acute causes of diaphragmatic or chest wall weakness, CVA, intracerebral haemorrhage, GBS	Horner's Syndrome, ALS, other progressive causes of diaphragmatic or chest wall weakness
Toxic/metabolic	Toxic gas exposure, acidaemia, salicylate overdose, opiate overdose, organophospate poisoning, DKA	
Haematological	Methaemoglobinaemia CO poisoning	Severe anaemia

Investigations

Testing should be based on history and physical findings.

- Labs: CBC (anaemia or infection), electrolytes, renal (acidosis, renal failure, electrolyte abnormality) ◇; D-dimer (PE), troponin (MI) ◈
- ECG ◇: (MI, arrhythmia, signs of toxin/poisoning)
- Imaging: CXR ◇ (pneumonia, PTX, pulmonary oedema, foreign body), VQ scan ◇; CT chest, echo ◈

Management

The goal of acute management is to restore oxygenation and ventilation, and identify and treat the cause. Specific management directed at the suspected aetiology.

Critical documentation

Serial VS, essential investigations and plan. Continually reassess unstable patients and document changes. Interventions and clinical response.

Disposition

Admit as per underlying condition.

240 Haemoptysis

Haemoptysis is a potentially life-threatening symptom that may indicate serious underlying pathology, and can range from blood-stained sputum to large volume haemorrhage. Causes range from mild tracheal irritation to pulmonary parenchymal destruction. Definitions of massive haemoptysis (< 2% of all cases with mortality ~80%) vary widely from 100–600 ml of blood over 24 hours. Any haemoptysis that causes anaemia or haemodynamic compromise should be considered massive/severe, regardless of the reported volume.

First five minutes

ABC, VS, O_2, IV, pulse oximetry, cardiac monitor

History and physical examination

Goals are to identify haemoptysis and localise source (tracheal, pulmonary, aspirated blood from epistaxis or GI source?), and to estimate amount and impact of blood loss.

- Distinguishing haemoptysis from haematemesis may be difficult for patients. Haemoptysis is usually bright, frothy and alkaline on testing
- Screen for signs of anaemia, haemodynamic compromise, respiratory distress
- Identify characteristic aetiologic features per table

Possible causes and differential diagnosis

The most common causes are acute bronchitis, TB, bronchiectasis and malignancy. The causes most likely to present with severe haemoptysis are TB, malignancy and vascular malformations. Life threatening conditions associated with often minor haemoptysis include PE and pulmonary oedema; no cause is found in 30% of cases.

Table 240.1 Causes of haemoptysis

Infections	Bronchitis, pneumonia, bronchiectasis, lung abscess, aspergilloma, TB
Malignancy	Upper airway or lower respiratory tract (primary and secondary); Kaposi Sarcoma
Trauma	Penetrating or blunt; foreign bodies
Cardiovascular	Pulmonary embolism, CCF, mitral stenosis, thoracic aortic aneurysm
Coagulation disorders	Anticoagulants (e.g. warfarin), thrombocytopaenia
Other	Cystic fibrosis, severe chemical pneumonitis, ateriovenous malformation, Goodpasture's syndrome, granulomatosis with polyangiitis

Extra pulmonary	Both ENT and GIT bleeding can be mistaken for haemoptysis

Investigations

- Labs: CBC, electrolytes, renal, type and cross blood if large volume. ◇ PT/PTT ◈
- Imaging: CXR ◇ (normal in about ⅓; may show infection, malignancy or chronic lung disease); CT (or CTA if considering PE) ◈
- Bronchoscopy ◈: (direct visualisation of the bronchial tree, collection of material for microscopy, culture and cytology, and therapeutic measures (e.g. balloon tamponade))

Management

The goal of acute management is to restore oxygenation and perfusion. Most cases are mild, secondary to infectious processes. However, patients with haemoptysis can deteriorate rapidly. Ensure early large-bore vascular access, antibiotics, cough suppression and close monitoring.

- Massive haemoptysis:
  - » Immediate threat is asphyxiation
  - » Advance ETT into the right main bronchus to protect the right lung if bleeding is suspected from the left lung
  - » If available, place a double lumen ETT to protect the normal lung
  - » Place patient with affected lung down (dependent)
- Mild-moderate: cough suppression is essential. Ongoing cough may increase injury and cause progression to severe haemoptysis

Critical documentation

Onset and progression, witnessed haemoptysis, interventions and clinical response.

Disposition

Discharge stable patients with small amounts of haemoptysis. Ensure TB testing and limit exposure of children and immunocompromised contacts. Admit all unstable patients, all moderate haemoptysis and those with ongoing cough; transfer/admit patients with massive haemoptysis to ICU care ◈.

241 Pleural effusion

Effusion is the accumulation of excess fluid in the pleural space, which normally contains < 20 ml of fluid. A pleural effusion is not a diagnosis: its presence mandates the search for an underlying cause. Pleural effusions occur due to:

- Transudate: disturbances in the hydrostatic-osmotic pressure gradient
- Exudate: pleural inflammation causing increased membrane permeability

The first five minutes

ABC, VS, O_2, pulse oximetry, cardiac monitor.

History and physical examination

- The aims are to identify effusion and its clinical manifestations (mediastinal shift, haemodynamic compromise, respiratory distress, empyema)
- The majority of pleural effusions are small and produce minimal disturbance to the patient
- May present with dyspnoea and often with mild discomfort on inspiration. A productive cough and haemoptysis suggest pulmonary pathology
- Chest pain may indicate malignancy, PE or pleural inflammation
- Ask about risk factors for malignancy (smoking, asbestos exposure) and TB
- Look for hypoxaemia. Clinical signs on affected side: Reduced chest wall expansion, stony dullness on percussion, reduced breath sounds, reduced vocal resonance
- Always consider empyaema: fever, moderate to severe chest pain, chest wall tenderness, and turbidity, pus,

organisms, or pH < 7.2 in pleural fluid aspirate

Possible causes and differential diagnosis

Congestive cardiac failure, TB, pneumonia and malignancy are the commonest causes of effusion. Pulmonary embolism is associated with effusion in many cases. Fluid analysis can distinguish transudate from exudate, which can help identify aetiology. (See below for criteria.)

Table 241.1 Causes and differential diagnosis of pleural effusion

Transudates		CCF, cirrhosis, nephrotic syndrome, peritoneal dialysis, hypothyroidism, hypoalbuminaemia, constrictive pericarditis, SVC obstruction, ovarian fibroma (Meigs' syndrome: rare)
Exudates	Infections	Bacterial and atypical pneumonia, TB, parasites, fungal disease, abscesses (subphrenic, hepatic, splenic, intra-abdominal), hepatitis
	Inflammatory	Pancreatitis, PE with infarction, ARDS, irradiation, pleuritus (uraemic, viral)
	Malignancy	Bronchial carcinoma, mesothelioma, lymphoma, leukaemia, chylothorax
	Other	Trauma, drugs (nitrofurantoin, dantrolene, cytotoxics), auto-immune disorders (rheumatoid arthritis, lupus), post-surgery

Investigations

All patients need a CXR and pleural fluid analysis (in patents with known CCF and bilateral effusions in the setting of acute exacerbation, consider fluid analysis only if effusion not resolved with CCF treatment). Other investigations should be guided by the suspected diagnosis.

- Labs: CBC, electrolytes, renal, type and cross (if unstable) ◇, PT/PTT ◈
- Imaging: CXR ◇ (PA/ lateral confirm and localise effusion > 250 ml); CT ◈ can often diagnose underlying cause
- Pleural aspiration ◇: fluid for microscopy, culture (including acid fast stain for AFB), cell counts (red blood cells, neutrophils, lymphocytes), cytology, biochemistry per below
- Light's criteria differentiate transudate and exudate. Exudate if:
  - » Pleural/serum protein ratio > 0.5
  - » Pleural/serum LDH ratio > 0.6
  - » Pleural LDH > 2/3 upper limit of normal for serum
  - » Bloody tap: TB or malignancy until proven otherwise

Management

The goal of acute management is to restore oxygenation and identify the cause of effusion. Treat the underlying cause. No specific treatment indicated unless causing hypoxia and/or significant dyspnoea; thoracentesis for diagnostic purposes and/or symptomatic relief (p. 836).

Critical documentation

Serial VS, all interventions and clinical response. After thoracentesis, document CXR or US evaluation for PTX.

Disposition

Discharge if small effusion in stable patient; admit hypoxic or dyspnoeic patients, and those with large or bloody effusions.

242 Spontaneous pneumothorax

A pneumothorax (PTX) occurs when air has collected between the two layers of the pleura. Air may enter the pleural space from the atmosphere (penetrating chest trauma and procedures), the airways/lungs, structures below the diaphragm (ruptured viscus), or gas-forming infections. Spontaneous PTX (as opposed to traumatic) may arise from structurally normal lungs. PTX can cause partial or complete lung collapse, resulting in VQ mismatch and hypoxia.

Tension PTX can develop when air enters the pleural cavity during inspiration but does not escape during expiration, resulting in progressive hyperinflation of the affected hemi-thorax. The greatest danger is mediastinal displacement with kinking of the great vessels, leading to reduced cardiac pre-load and decreased cardiac output.

The first five minutes

- ABC, VS, O_2, pulse oximetry, cardiac monitor
- Identify tension PTX (hypotension or poor perfusion, dilated neck veins, hypoxia, and (rarely) palpable deviation of the trachea away from affected side) and treat:
  - » Large IV cannula to 2nd intercaostal space, mid-clavicular line on the affected side

History and physical examination

Key historical features

- Pre-existing lung pathology, e.g. asthma or COPD, TB (most common cause is ruptured pulmonary bullae)
- Other significant co-morbidities
- History of smoking tobacco, cannabis or metaqualone (mandrax) (significantly associated with PTX)
- Significant dyspnoea (usually sudden)

Signs and symptoms

- Asymmetrically reduced or absent breath sounds
- Reduced vocal resonance
- Asymmetrical hyper-resonant percussion note
- Subcutaneous emphysema indicates an air leak that may be from the lung, major airways or oesophagus

Differential diagnosis

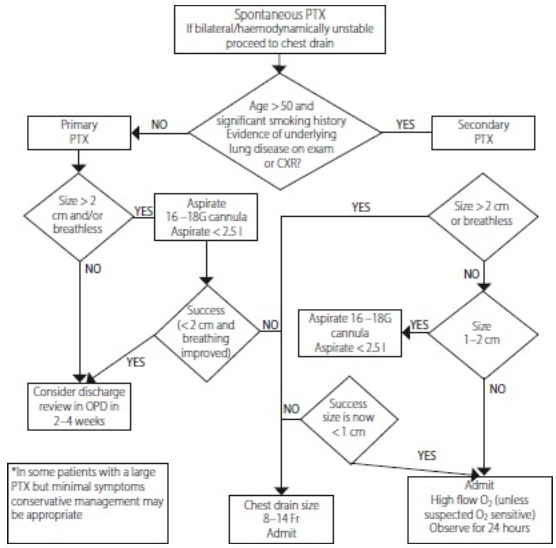
Musculoskeletal chest pain, PE, chest infections, aortic dissection, ACS.

Investigations

- Imaging: CXR (erect and inspiratory usually make definitive diagnosis; excludes some of the differential; expiratory films are useful for small PTX); US (good sensitivity and specificity) (Ultrasound, p. 787, Trauma, p. 742, and Dyspnoea, p. 630) ◇

Management

The goal of acute management is to restore oxygenation.



From: Macduff A, et al. 2010. Management of spontaneous pneumothorax: British Thoracic Society pleural disease guideline 2010. *Thorax*, 65(Suppl 2):ii18–ii 31.

Disposition

Avoid air travel until full resolution. Avoid diving permanently.

Admit if significant underlying lung disease (COPD, pulmonary fibrosis, infections); co-morbidity (CV disease, etc.); previous PTX, or chest tube in place.

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Part

4

Q. Rheumatology

243 Approach to arthritis

Arthritis refers to inflammation of one or more joints.

This chapter should be read in conjunction with the chapter on acute monoarthritis (📖 p. 642).

History and physical examination

Key historical features

- Onset, character, distribution and duration of joint symptoms (pain, redness, swelling, heat, loss of function). Morning stiffness and improvement (more likely inflammatory) or aggravation (more likely degenerative) with activity
- Presence of associated extra-articular or constitutional symptoms; ask about any genito-urinary symptoms (gonococcal or reactive arthritis)
- History of trauma, recent infection, joint prosthesis, IV drug abuse, or indwelling vascular lines or devices
- Family history of auto-immune and joint diseases
- Distribution:
  - Mono (one joint); oligo (few joints); poly-arthritis (many joints)
  - Large vs. small joints and symmetrical vs. asymmetric
  - Single, large, asymmetrical joint involvement: gout, infection, degeneration
  - Multiple, small symmetrical joint involvement: auto-immune reactive

Signs and symptoms

The four most important presenting patterns are:

- Mono- or oligo-arthritis with fever and evidence of systemic illness: septic arthritis until proven otherwise, but also consider gout
- Symmetrical small joint poly-arthritis with a sub-acute or chronic course associated with systemic signs and symptoms: probable auto-immune (RA)
- Involvement of large weight bearing joints with a chronic course in older patients: consider degenerative conditions (OA)
- Unexpected arthritis following any infectious disease with spontaneous resolution: probable reactive arthritis

Look for: fever, distribution, joint redness and heat, joint swelling (synovial – boggy, or bony – hard), joint effusions and range of movement. Remember to search for associated systemic signs.

Redness, swelling, and effusion are difficult to appreciate at less accessible joints (e.g. hip): pain, joint irritability,

and limited range of motion are often the main findings.

Possible causes and differential diagnosis

The most important distinction is that between septic and non-septic arthritis as delayed treatment of the former greatly increases tmorbidity and mortality.

- Septic arthritis (📖 p. 644)
- Gonococcal: typically large joint oligo-arthritis in young adults. May have a migratory pattern. Often systemically unwell; palmar or plantar rash
- TB: always consider in mono- or oligoarthritis
- Reactive arthritis: autoimmune inflammation triggered by an infection elsewhere in the body. Usually transient
- Transient synovitis: short-lived acute joint inflammation, usually between 2 - 10 years old, often following an upper respiratory tract infection
- Gout and pseudogout (📖 Acute mono-arthritis, p. 642)
- Rheumatoid arthritis: chronic autoimmune inflammation of the joints of unknown aetiology, often associated with systemic symptoms. First presentation is often acute, single joint
- Osteoarthritis: inflammation from mechanical forces of overuse, aging, 'wear and tear' of the joints, or as the sequelae of musculoskeletal injury
- Rheumatic fever: inflammation resulting from inadequately treated group A beta-haemolytic streptococcal pharyngitis (📖 p. 370)
- Traumatic effusion

Investigations

Consider bloodwork in systemically unwell patients. Perform arthrocentesis if:

- First presentation
- Large joint involvement
- Fever
- Systemically unwell
- Evidence of infection in the areas surrounding the joint (📖 Arthrocentesis, p. 852)

Management

The goal of acute management is to relieve symptoms and find and treat the cause. For septic arthritis and gout see 📖 Acute mono-arthritis, p. 642). Weight loss important in degenerative arthritis. When auto-immune arthritis is suspected – early specialist referral for initiation of disease modifying antirheumatoid drugs (DMARDs).

Symptomatic treatment

- Paracetamol and NSAIDs (oral or topical)
- Opiates for breakthrough pain – avoid chronic use
- Compression bandage and crutches as needed for comfort

Disposition

Admit septic arthritis. Most other patients can have out-patient management.

244 Acute mono-arthritis

Arthritis refers to inflammation of one or more joints. This chapter should be read in conjunction with the approach to arthritis chapter (📖 p. 640). Acute mono-arthritis is an important presentation because it may reflect a potentially devastating diagnosis (septic arthritis), or a very common and debilitating condition (gout).

Possible causes and differential diagnosis

Mono-arthritis is usually caused by:

- Septic arthritis (including gonococcal)
- Crystal arthropathies (gout, pseudogout)
- Degenerative joint disease with an acute inflammatory flare

Also consider trauma, reactive arthritis, atypical auto-immune arthritis, soft tissue infections surrounding the joint (bursitis, cellulitis) and tumours (Ewing sarcoma, osteosarcoma).

History and physical examination

Usually presents with a painful joint and inflammation (swelling, redness, heat and joint effusions). Ask about pre-existing joint disease, previous joint injury or trauma. Fever and malaise may indicate septic arthritis. Weight loss, constitutional symptoms and a more chronic course may indicate TB or malignancy (particularly knees and elbows of teenagers and young adults).

Septic arthritis

Intense joint inflammation, significant pain (even on passive joint manipulation) and systemic illness (fever, malaise, etc.). Usually caused by *Staph* spp. and *Strep* spp. Other causes include gonococcus (often oligo-arthritis associated with typical skin lesions and an STI) and TB. More common in large joints, children and structurally abnormal or prosthetic joints. Affects the knee in 50%. Carefully examine the surrounding soft tissues for draining sinuses.

Gout

Intense joint inflammation due to deposition of urate crystals in the joint space. Associated with hyperuricaemia. More common in men; incidence increases with age. Most commonly involves: first metatarsal-phalangeal joints, base of the thumb, elbow and knee (but any joint may be involved). Chronic gout is associated with joint destruction and the formation of cutaneous tophi and renal stones. In severe cases, gout may cause fever. Joint destruction from chronic gout increases risk of septic arthritis.

Pseudogout

Inflammation from calcium pyrophosphate crystals in the synovial fluid. More likely to involve large joints, primarily the knee.

Investigations

Arthrocentesis: see p. 852

- White blood cell count ≥ 25 000/µl or PMN cell percentage > 90% suggests septic arthritis. If no lab, fluid that is grossly opaque with decreased viscosity and any colour other than straw – treat as septic
- Do not measure serum uric acid during an acute attack of gout – levels are falsely low and may lead to the inappropriate exclusion of the diagnosis

Management

The goal of acute management is to relieve symptoms and find and treat the cause. Advise elevation and ice packs.

Septic arthritis

- Early specialist referral for joint irrigation in OT; sterile irrigation and drainage by arthrocentesis if referral not available
- Parenteral antibiotics 14–21 days, then oral 14 days
- Gram negative bacilli coverage – elderly or history of IV drug use or immunocompromise; cephalosporins if gonococcus suspected

Gout

- NSAIDs are first line treatment. Avoid aspirin
- If not effective, consider: corticosteroids (PO or intra-articular – exclude infection first); colchicine (0.5 mg PO hourly to a max of 8 mg, but almost always causes diarrhoea and vomiting)
- Long term uric-acid lowering therapy should be considered once acute attack resolved. Do not start, stop or change the dose of allopurinol during an acute attack

Pseudogout

- NSAIDs are first line treatment

Disposition

Admit septic arthritis. Most patients with gout can be discharged. Arrange specialist review for gout associated with joint destruction or renal disease, and if you suspect TB arthritis.

245 Systemic lupus erythematosus

Systemic lupus erythematosus (SLE) is a chronic auto-immune illness of unknown cause that may affect any organ and has a variable clinical course. Common initial features include joint pain and myalgia, skin changes and malaise. The dominant early clinical syndrome in an individual patient typically persists throughout the illness. Young adult women are most commonly affected.

Diagnosis is rarely made in the emergency setting. Patients with SLE will often present with multiple vague constitutional symptoms that can lead to recurrent and frustrating visits before the disease is correctly diagnosed.

History and physical examination

Patient presentations are not predictable and SLE mimics other autoimmune conditions, infectious diseases and malignancies. SLE usually begins with one or more of the following:

- Constitutional symptoms (fever, fatigue, weight loss)
- Photosensitive rash (raised, painless, erythematous butterfly malar rash is classic)
- Arthralgia or arthritis, often symmetrical involvement of the small joints
- Nephritis
- Neurologic or psychiatric symptoms
- Pleuritis, pericarditis, or peritonitis
- Recurrent miscarriage
- Anaemia

SLE should also be suspected in young women presenting with purpura, easy bruising, unexplained rashes, especially of the face, unexplained arthralgia and arthritis, diffuse adenopathy, hepatosplenomegaly, peripheral neuropathy, endocarditis, myocarditis, interstitial pneumonitis, or aseptic meningitis.

Differential diagnosis

As SLE can present in numerous ways. The differential diagnosis is very broad and includes:

- Antiphospholipid syndrome
- Fibromyalgia
- Infectious mononucleosis
- Infective endocarditis
- Lymphoma, B-Cell
- Mixed connective tissue disease
- Polymyositis
- Rheumatic fever
- Rheumatoid arthritis
- Scleroderma

Investigations

Testing should be guided by the clinical presentation.

- Labs: CBC, electrolytes, renal, ESR; urinalysis ◇; CRP ⟐. Autoantibodies: antinuclear antibodies (ANA), anti-double stranded DNA (dsDNA) and anti-ribosomal (RNP) ⟐
- Imaging: as directed by clinical findings

Management

The goal of acute management is recognition in the undiagnosed patient, reduction of inflammation, symptomatic management during acute flare-ups and early referral to a specialist.

Immunosuppresive drugs are the cornerstone of therapy and include glucocorticoids, and disease modifying antirheumatic drugs (DMARDs, such as methotrexate, chloroquine and gold).

Emergency treatment is tailored to the type and severity of the presenting symptom.

Once diagnosed, patients may be treated with a variety of medications that are used to mitigate inflammation; many of these therapies have unacceptable side effect profiles in chronic use.

A well patient with suspected SLE is best served with symptomatic treatment and referral to a specialist. Unwell patients should receive symptomatic management, emergency stabilisation of deranged physiology and urgent admission to a specialised in-patient service.

Disposition

Primary care and/or internal medicine follow-up is essential. Acutely unwell patients should be admitted to internal medicine.

246 Approach to vasculitis

Vasculitis is a term for a large group of disorders characterised by inflammation of blood vessels and may be caused by drugs, infections or auto-immune processes. It is rare, but has the potential to threaten life, organ and limb. Patients usually present with vascular abnormalities and varying systemic inflammatory features ranging from slight malaise to severe illness. Other auto-immune and inflammatory phenomena, such as involvement of the joints, skin, mucosa and serosae are common.

Vasculitides are commonly divided into three groups based on the predominant vessel size involved.

Clinical features

Often a prodromal phase with non-specific constitutional symptoms, followed by more specific systemic abnormalities (particularly arthralgia and malaise) and compromise of vascular integrity.

Large vessel vasculitides

Takayasu’s arteritis (TA)

Mainly involves the aorta and its branches; rare > 50 years. Causes bruits, absent or asymmetric pulses, and claudication. > 50% have hypertension (from renal artery stenosis). Diagnosis is based on typical clinical and radiologic findings.

Giant cell arteritis (GCA)

Predominantly involves the cranial branches arising from the aorta; rare < 50 years. Symptoms include new onset unilateral headache, jaw claudication, visual abnormalities and tenderness of temporal artery. Diagnosis is based on clinical findings and a raised ESR. Biopsy should be performed but should not delay treatment (delay may cause irreversible blindness). Commence high dose steroids.

Medium vessel vasculitides

Polyarteritis nodosa (PAN)

Rare; usually idiopathic, but may be associated with Hep B, Hep C, or hairy cell leukaemia. Often results in arterial aneurysms that may rupture or thrombose. Presents with systemic features; diagnosis confirmed with biopsy. If unable to biopsy, angiography may reveal micro-aneurysms in various organs.

Kawasaki's disease (KD) 📖 p. 367

Mainly occurs in children; associated with a syndrome of rashes and lymph node reactivity (typically bilateral non-exudative conjunctivitis, erythema of the lips and oral mucosa, rash, extremity changes, and LAN). See 📖 Kawasaki p. 367 for criteria and treatment. May be complicated by coronary artery aneurysm and peripheral arterial occlusion.

Syphilitic arteritis

Affects aortic arch; still common in Africa.

Basal CNS arteritis

Commonly associated with TB meningitis; can lead to posterior circulation strokes and bleeds.

Small vessel vasculitis

Eosinophilic granulomatosis with polyangiitis (EGPA)

Formerly called Churg-Strauss disease; classically involves arteries of the lung and skin. Prominent eosinophilia. Diagnosis is based on clinical findings and biopsy results. Anti-Neutrophil Cytoplasmic Antibody positive ⟐.

Granulomatosis with polyangiitis (GPA – formerly Wegener's granulomatosis) and microscopic polyangiitis (MPA)

Present with constitutional symptoms, upper respiratory (ENT, larynx), lower respiratory (wheezing, granulomas) and renal findings. Mostly occurs in adults. Differences are based on histologic findings. Diagnosis is based on a positive ANCA and biopsy findings ⟐.

Henoch-Schönlein purpura (HSP)

Classically presents with a tetrad of abdominal pain, arthralgia, renal involvement and purpura (in dependent areas and in the absence of thrombocytopenia or coagulopathy). Diagnosis is clinical and biopsy is reserved for atypical presentations. Evaluation of renal function is essential. Treatment is generally supportive with hospitalisation reserved for severe symptoms. Steroids are reserved for those patients with severe abdominal pain (after ruling out other causes) and significant renal involvement.

Differential diagnosis

The differential is very broad and incudes infections, scurvy, sickle cell disease, TTP, antiphospholipid syndrome, Hep B, Hep C, HIV.

General diagnostic approach

After initial symptomatic treatment and diagnostic work-up, early involvement of a specialist is essential.

Exclude other processes that may mimic vasculitis

Important to avoid misdiagnosis as steroid treatment for vasculitis may exacerbate infectious conditions.

Exclude secondary cause

Most secondary vasculitides are extremely rare and readily diagnosed by features of the parent illness (inflammatory disease, infectious diseases, neoplasia and almost any drug – take a complete drug history (cessation of the drug

leads to resolution of the vasculitis)).

Determine the distribution and extent of vasculitis

A thorough history and physical may yield clues. Evaluate and document ALL pulses and the perfusion of the peripheries. Evaluate renal function, and perform urine dipstick. Haematuria necessitates microscopy to determine the presence of casts. CXR (pulmonary involvement in otherwise asymptomatic patients).

Confirm the diagnosis – histologic and radiographic confirmation

- Clinical examination should be focused on identifying a suitable site for biopsy. Blind biopsy to exclude vasculitis is unhelpful
- Renal function tests in patients with haematuria or proteinuria. Inflammatory markers little use (raised in primary vasculitis, mimics, and secondary causes). Serum ANCA levels ◈ highly suggestive of small vessel vasculitis (negative ANCA does not exclude vasculitis)
- Tissue diagnosis is impractical in large to medium vessel vasculitides; order angiography ◈ of the appropriate vessels (look for stenosis, occlusion and aneurysms)

Determine the specific type of vasculitis

The clinical context, labs and imaging should guide the clinician towards a diagnosis. Observation over time with repeat investigations and a therapeutic trial may increase or decrease the diagnostic certainty.

Management

The management of vasculitis is dependent upon the diagnosis, the nature of the symptoms and extent of organ involvement. Treatment options include observation alone, steroids alone or in combinations with disease modifying agents or invasive radiologic procedures. Consult a specialist early.

Disposition

Admit or refer to a specialist physician or rheumatologist.

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Part

4

R. Toxicology

247 Approach to the acutely poisoned patient

Acute poisoning can occur by ingestion, inhalation, injection, or cutaneous or mucosal absorption. Exposure may be intentional (suicide or self-harm) or unintentional (e.g. snakebite, medication error, and environmental or

occupational exposure). Overdose victims include body stuffers (persons who quickly swallow drug packages in an effort to hide evidence from law enforcement) and body packers (persons who seal large amounts of drugs in packages and then swallow them for transport).

All substances are potentially toxic, depending on the dose and route of exposure. Knowledge of the agent involved is important to predict, prevent and/or manage clinical effects, but often the specific substance will not be identified. Antidotes exist for only a limited number of poisons and may not be widely available.

The first five minutes

- Terminate the exposure: remove clothes and wash skin with simple soap if there has been exposure to liquid chemicals (caustics, pesticides, etc.). If the skin or eyes affected, immediately flush with plain water or saline. Use activated charcoal when indicated in recent ingestions in alert patients
- ABC, VS, IV access, O_2 for hypoxia, cardiac monitor
- Assess mental status, measure glucose if altered

History and physical examination

Key historical features

- Determine specific agent and dose: ask family to bring suspect containers or samples for inspection in unknown cases. Photographs of containers/labels can be helpful
- Probe circumstances: cause and/or reason for exposure (e.g. unintentional, intentional, suicide attempt, recreational, occupational, therapeutic)
- Ask about any pre-hospital care given (e.g. decontamination measures)
- Consider common sources of exposure to potential toxins in air, food, medication, water, or manufactured products if more than one patient presents with similar symptoms (e.g. carbon monoxide poisoning)

Signs and symptoms

- Examine thoroughly for signs of trauma, stigmata of chronic illness
- Look for clues to specific poisons: odours, content and colour of vomitus, burns, puncture marks suggestive of snake or scorpion bite, self-injection
- Check VS, pupils, skin, bowel, and bladder function to look for toxidromes

Table 247.1: Toxidromes

Toxidrome	Clinical features
Anticholinergic (p. 690)	Tachycardia, hyperthermia, agitation, delirium, mydriasis, dry flushed skin, urinary retention
Cardiodepressant (p. 694)	Hypotension, bradycardia
Cholinergic	Vomiting, diarrhoea, excess salivation, lacrimation, urinary incontinence, miosis, bronchorrhoea, bronchospasm, bradycardia, muscle fasciculation, weakness
Opioid (p. 662)	Deep sedation, miosis, respiratory depression
Salicylates (p. 658)	Rapid breathing, tinnitus, altered mental status, metabolic acidosis
Sedative-hypnotic (p. 672)	Coma with normal or depressed VS (moderate hypotension, bradycardia)
Sympathomimetic (p. 692)	Tachycardia, hypertension, hyperthermia, agitation, diaphoresis, mydriasis

Differential diagnosis

- Consider a broad list of drugs, poisons and medical conditions
- Rule out hypoglycaemia in any altered patient
- Consider CNS infection in any patient with fever and ALoC

• Consider CNS trauma

Investigations

Choice of investigations is determined on a case-by-case basis.

• Labs: CBC, elec, renal, urinalysis, LFT, PT/PTT
  » Quantitative tests for specific poisons ◈ may help guide management decisions in select circumstances (e.g. paracetamol, iron, lead, digoxin, etc.)
  » Urine drug screening ◈ is rarely useful as most cases can be suspected on the basis of clinical presentation
  » Calculate osmolality
• ECG ◇: can help diagnose or gauge poisoning severity. Look for QTc prolongation, digoxin effect, signs of tricyclic antidepressant poisoning, ectopy, dysrhythmias

Management

ABC: ensure patent airway, intubate trachea and assist ventilation if needed (◇/◈). IV and bolus saline 1–2 l (adults) or 15–20 ml/kg (children)

Give IV dextrose bolus if hypoglycaemic.

Gastrointestinal decontamination is of uncertain benefit, especially if ingestion more > 1 hour before treatment:
  » Consider gastric lavage only if the patient presents soon after a potentially severe ingestion. Avoid in case of caustic ingestion
  » Forced emesis increases the risk of aspiration and should be avoided
  » Cathartics and/or forced diuresis have no proven benefit: avoid
  » Activated charcoal (AC) 1 g/kg may be given to help reduce absorption of ingested poisons ◇. AC does not bind alcohols, caustics, hydrocarbons, iron, or sodium, potassium, magnesium, lithium salts. Pulmonary aspiration is a major risk in lethargic patients: do not give AC routinely and avoid if the patient has altered consciousness or impaired protective airway reflexes
• Control seizures or extreme agitation: diazepam 5–10 mg IV (repeat dose in five minutes if no response) or other benzodiazepine is the first-line choice of medication for seizure control in poisoned patients. Consider pyridoxine (B6) if isoniazid (INH) toxicity is a possible cause ◇
• Antidotes may be life-saving in select cases; check with your pharmacist and formulary ahead of time to know what you may have available

Critical documentation

• Specific agent, dose, and time of ingestion or exposure (if known)
• Any pre-hospital care given (e.g. decontamination measures)
• Cause and/or reason for ingestion (e.g. accidental, intentional, suicide attempt, recreational, therapeutic)
• Determine if other family members or co-workers were also exposed if environmental or occupational exposure suspected
• Note if case was reported and where (e.g. to poison centre or other health unit)

Disposition

• Admit patients with moderate to severe symptoms or signs. In most cases, patients with minimal or no symptoms/signs can be discharged after about six hours of observation
• Psychiatric and/or social work evaluation if poisoning was intentional
• Counsel discharged patients and family to return in case of new or worsened symptoms
• Public health officials should investigate the site or source of an environmental, occupational, or product-related exposure to prevent others from being poisoned

248 Paracetamol (acetaminophen) poisoning

A widely used analgesic-antipyretic, and a component of many over-the-counter and prescription medications. Overdose is very common and toxicity is easily underestimated.

The first five minutes

- ABC, VS, facemask O_2, cardiac monitor
- Ensure adequate ventilation

Identify toxic dose

- Single dose: children > 150 mg/kg, adults > 7.5 g
- Repeated doses: children > 150–175 mg/kg over 2 to 4 days; adults > 12 g in 24 hours
- High risk patients > 100 mg/kg
- Intravenous overdose > 60 mg/kg

History and physical examination

Key historical features

Time and amount of ingestion. Any co-ingestions. Intent: suicide, accidental, therapeutic.
High-risk factors

- Glutathione deficiency: chronic alcoholism, malnutrition, starving/anorexia, HIV, acute illness, malignancy and acute hepatitis
- Medications and co-ingestants inducing liver enzymes: barbiturates, carbamazepine, phenytoin, rifampicin etc.

Signs and symptoms

- Stage 1 (0.5 to 24 hours): asymptomatic, nausea, vomiting, abdominal pain, diaphoresis and malaise
- Stage 2 (24 to 72 hours): right upper quadrant pain; liver enlargement and tenderness. Elevated serum transaminases (ALT, AST), prothrombin time, and total bilirubin. Oliguria and renal function derangements can occur
- Stage 3 (72 to 96 hours): jaundice, hepatic encephalopathy and bleeding diathesis with marked elevation in hepatic enzymes, hyperammonia. Severe hepatotoxicity indicated by confusion, lactic acidosis, hypoglycaemia, ALT and AST > 10 000 IU/L, elevated INR or PT, and total bilirubin > 4 mg/dl. Most deaths occur in this stage due to multi-organ failure
- Stage 4 (4 days to 2 weeks): recovery phase in survivors

Differential diagnosis

Medical conditions causing hepatic dysfunction: alcoholic hepatitis, drug- or toxin-induced hepatitis, viral hepatitis, hepatobiliary disease, Reye’s syndrome.

Investigations

- Labs: preganancy test (paracetamol crosses the placenta – high abortion risk in first trimester) ◇; acetaminophen level (all patients – at least four hours after acute oral ingestion (two hours for IV overdoses)); repeated levels in sustained release products or chronic supratherapeutic exposures), LFT, PT/PTT ◈.

Management

The goal of acute management is to reduce absorption if early (single dose activated charcoal if no contra-indications and < 1 hour since ingestion), and to minimise the toxic effects of byproducts of paracetamol metabolism.

Acetylcysteine

Indications:

- Appropriately timed paracetamol level plotted above the treatment line on the modified Rumack-Matthew nomogram (Figure 248.1)

• Suspected toxic overdose (do not delay treatment while awaiting levels; stop acetylcysteine if level below treatment line)
• Measurable paracetamol level or hepatotoxicity in patients presenting after 24 hours
• Abnormal LFTs and measurable paracetamol level in repeated supratherapeutic exposure (it is never too late to give acetylcysteine)
• Acetylcysteine is safe in pregnancy

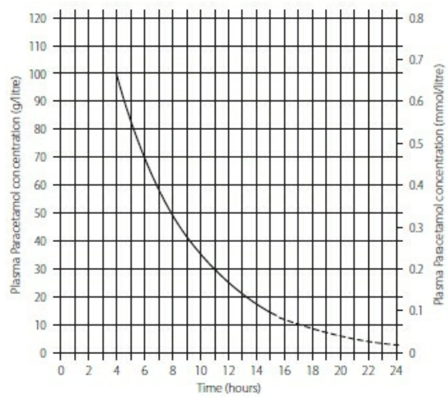

Figure 248.1 The modified Rumack-Matthew nomogram

Dose
• IV: initially 150 mg/kg in 200 ml 5% dextrose over 60 min (max 15 g); then 50 mg/kg in 500 ml 5% dextrose over the next four hours (max 5 g); followed by 100 mg/kg in 1 litre 5% dextrose over 16 hours (max 10 g)
• Oral: loading dose of 140 mg/kg followed by 70 mg/kg every four hours for a total of 17 doses (use actual body weight)
• Continue until paracetamol level is undetectable, the ALT within the normal range or clearly decreasing, and the INR < 2

Adverse effects
• Nausea and vomiting: anti-emetics (e.g. ondansetron) are useful. Repeat oral dose if patient vomits within 60 minutes
• Anaphylactoid reaction: severity ranges (urticaria to respiratory distress and cardiovascular collapse). Stop acetylcysteine and treat allergic reaction. Restart if symptoms settle. Hypotension or persistent systemic symptoms: switch to oral regime

Critical documentation

Amount ingested, single or repeated doses, time of the ingestion, and whether high risk. Any co-ingestants, and pre-existing comorbid conditions.

Disposition

Discharge patients with appropriately timed serum concentrations below the treatment line. Admit all others.

249 Salicylate (aspirin) poisoning

Aspirin is a widely prescribed analgesic and anti-platelet agent whose main ingredient is salicylate, which is also found in other products, e.g. methyl salicylate (oil of wintergreen) and salicylic acid (wart remover). Salicylate intoxication (salicylism) may be acute, chronic, or acute-on-chronic, and may occur via ingestion or topical therapies. Salicylates eventually cause a significant metabolic acidosis with uncoupling of oxidative phoshorylation and accumulation of organic acids (with only a small contribution by the salicylate itself). However, because there may be overlapping and progressive phases of acid-base disturbance – respiratory alkalosis (hyperventilation resulting from direct respiratory centre stimulation) and progressive metabolic acidosis – a normal serum pH does

not rule out significant toxicity.

The first five minutes

- ABC, VS, O_2, pulse oximetry, cardiac monitor
- Recognise hyperpnoea (including increased depth of breathing with or without tachypnoea)

History and physical examination

Key historical features

Include as much detail about the ingestion as possible. Note that chief complaint may not be for ingestion, but for a condition (e.g. headache) that patient is treating with aspirin. Possible fatal ingestion: adults > 10 g and children > 3 g.

Signs and symptoms

- Early (< 2 hours): nausea, vomiting, diarrhoea, hyperpnoea, tinnitus, vertigo
- Severe intoxication: hyperpyrexia, hypovolaemia, altered mental status (agitation to lethargy), non-cardiac pulmonary oedema, dysrhythmias, seizures and coma
- Clinically significant bleeding is uncommon

Investigations

- Labs: electrolytes (treat hypokalaemia aggressively), renal (renal failure is an absolute indication for haemodialysis), glucose, ABG (various acid-base disturbances can occur: respiratory alkalosis, metabolic acidosis, respiratory acidosis) ◇; serum salicylate (six hours post ingestion; expect toxicity > 40 mg/dl (2.9 mmol/L); repeat every two hours until it is declining), serum lactate (elevated in significant salicylate poisonings) ⟐
- Imaging: CXR ◇ (if pulmonary oedema present)

Differential diagnosis

- Consider other toxicological causes (iron, ethylene glycol, hydrocarbons, theophylline organophosphates/carbamates and withdrawal syndromes)
- Consider other medical causes (diabetic ketoacidosis, sepsis, pneumonia)

Management

The goal of acute management is decreased absorption early in acute overdose, alkalinisation of serum and urine to decrease CNS exposure and increase excretion, aggressive management of electrolyte disturbances, and supportive care for CNS and cardio-respiratory effects.

- Administer supplemental O_2 as needed but avoid intubation if possible. Patients with severe toxicity may require intubation for oedema and decreased oxygenation, but tolerate it very poorly, as they are often dependent on high respiratory rate to maintain acid-base balance. Prepare for peri-intubation haemodynamic instability
- Gastric lavage ◇ and activated charcoal if recent and no contra-indications
- IVF to treat dehydration and maintain urine output (1–1.5 ml/kg/hr). Note: CAUTION for pulmonary oedema
- Correct hypoglycaemia and electrolyte disturbances ◇ (acidosis may mask hypokalaemia)
- Alkalinisation of serum and urine is essential in all symptomatic patients ◇. Sodium bicarbonate bolus (1–2 mEq/kg), followed by an infusion (100–150 mEq in 1 litre D5W) to a urine pH of 7.5–8. Correct hypokalaemia before administration
- Hyperthermia managed by external cooling, not anti-pyretics
- IV benzodiazepine for seizures
- Haemodialysis ⟐: indications include serum level > 100 mg/dl (7.2 mmol/l) six hours post ingestion, refractory acidosis, coma or seizures, non-cardiogenic pulmonary oedema, volume overload, and renal failure

Critical documentation

- Note local units for all serum levels
- Note presenting and progressive signs and symptoms, and response to interventions
- Serial ABG, especially in intubated patients

Disposition

- Admit all symptomatic patients, individuals with chronic salicylism and patients who ingested sustained-release products
- Admit severe poisonings to ICU
- Observe asymptomatic patients for six hours before discharge

250 Non-steroidal anti-inflammatory drugs

Non-steroidal anti-inflammatory drugs (NSAIDs) include more than 20 drugs broadly classified into carboxylic acid, enolic acid derivatives and COX-2 selective inhibitors. They all share the ability to inhibit prostaglandin (PG) synthesis and have analgesic, anti-pyretic and anti-inflammatory effects. NSAIDs are among the most commonly used medications in the world. Common NSAIDs include ibuprofen, diclofenac, indomethacin, ketorolac and naproxen.

Significant toxicity and death are rare after NSAID-only overdose.

The first five minutes

- ABC, IV for symptomatic patients, large ingestions (e.g. deliberate self-harm, child with > 400 mg/kg)
- CNS depression may warrant airway protection with positioning, nasal/oral airway or ETT
- Cardiac monitoring (if available). Look for signs of hyperkalaemia
- Check glucose if AMS, lethargy, seizure

History and physical examination

Key historical features

Time and amount of ingestion. Any co-ingestions. Intent: suicide, accidental, therapeutic.

Signs and symptoms

- Most patients develop mild GI symptoms – nausea, vomiting, abdominal pain
- Minor CNS symptoms such as drowsiness, dizziness, headache, diplopia, CNS depression and tinnitus may also occur
- More severe: seizures, hypothermia, bloody emesis, GI haemorrhage, oliguric renal failure, metabolic acidosis and rhabdomyolysis
- Monitor mental status: CNS depression is usually mild but may progress to delirium or coma
- Chest examination: fast, shallow respiration may be compensation for metabolic acidosis in severely poisoned patient. Examine for crepitation/crackles since patients with vomiting and/or depressed consciousness are at high risk of aspiration
- Meningeal signs (Kernig's and Brudzinski's sign) may be present with NSAID-induced aseptic meningitis

Differential diagnosis

- Abdominal pain, vomiting, and/or gastrointestinal bleeding should prompt consideration of alcohol-induced, infectious, uremic, or other gastritis, iron or other heavy metal poisoning, ingestion of a caustic/irritant
- Meningitis and encephalitis should be considered in all patients with neurologic signs
- Hypoglycaemia should be considered in any altered patient

Investigations

Drug concentrations not usually useful, and do not correlate with symptom severity.

- Labs: CBC, electrolytes, renal, ABG, LP (patients with AMS or meningeal signs, to rule out infectious aetiology; in NSAID-induced aseptic meningitis, CSF typically shows mild pleocytosis, an elevated total protein and normal glucose level) ◇

Management

The goal of acute management is supportive care, protection of airway in altered patients, and treatment of associated GI bleed.

- Treatment is largely supportive and based on clinical condition
- Good IV access is crucial as patients may develop life-threatening GI haemorrhage and require urgent transfusion
- Gastrointestinal decontamination with activated charcoal ◇ may be useful in case of recent acute overdose. Gastric lavage is of unproven benefit and may provoke vomiting and pulmonary aspiration
- Proton pump inhibitors are indicated for NSAID-induced gastritis/ulceration ◇

Critical documentation

Time of exposure, dose, and type of preparation taken. Other co-ingestants particularly in the setting of a deliberate ingestion.

Disposition

Discharge asymptomatic patients with normal VS after four hours of observation, and children with a reliable history of ingesting less than 100 mg/kg BW may be observed at home. If more than 400 mg/kg BW, admit. Also admit symptomatic patients.

251 Opioid analgesics

Opioids include codeine, buprenorphine, morphine, diamorphine (heroin), pethidine and methadone. Commonly used recreationally; overdose victims include body stuffers (persons who quickly swallow drug packages in an effort to hide evidence from law enforcement) and body packers (persons who seal large amounts of drugs in packages and then swallow them for transport).

The first five minutes

- ABC, VS, facemask O_2, continuous pulse oximetry
- Ensure adequate ventilation

History and physical examination

Key historical features

Timing and amount of ingestion. Any co-ingestions? Intent: suicide, accidental, therapeutic. Stuffing or packing? Any long-acting (slow-release) formulations? Any naloxone given pre-hospital?

Signs and symptoms

- CNS: depressed level of consciousness, pinpoint pupils (normal pupil size does **not** exclude opioids), and seizures. Excitatory effects (hallucinations, tremors, muscle twitches and dilated pupils) may be seen with pethidine toxicity
- Respiratory: decreased respiratory rate with shallow respiration or apnoea, cyanosis. Coarse crackles may indicate non-cardiogenic pulmonary oedema (heroin), aspiration or acute lung injury

- GIT: decreased bowel sounds
- CVS: hypotension with normal to low pulse rate
- Hypothermia (fever may indicate aspiration pneumonia or septic complications from injections); measure core temperature ◇

Differential diagnosis

- Any medical condition that can result in a depressed LoC (hypoglycaemia, meningitis, diabetic ketoacidosis etc.)
- Other toxicological causes of a depressed LoC (alcohol, anti-depressants, sedatives-hypnotics etc.)

Investigations

Opioid toxicity is a clinical diagnosis. No need for routine labs or urine toxicology screens.

- Imaging: CXR (if pulmonary complications suspected), AXR (when evaluating a suspected body stuffer or body packer) ◇

Management

The goal of acute management is airway protection in altered patients and identification and treatment of respiratory depression.

- Ensure patent airway and adequate ventilation
- Single dose activated charcoal only for patients with life-threatening co-ingestants
- Naloxone is an opioid antagonist. Initial adult dose: 0.4–2 mg IV or IM. If no improvement occurs, repeat dose at 2–3 minute intervals up to max 10 mg. Huge heroin overdose may require a 0.004 mg/ml naloxone infusion (dilute 2 mg of naloxone in 500 ml of 0.9% saline or 5% dextrose solution). In children, the initial IV dose is 0.01 mg/kg followed by 0.1 mg/kg, if necessary. The duration of action of naloxone is shorter than most opioids so respiratory depression will recur. Patients should be observed carefully as repeated doses may be required after the initial improvement. The goal of naloxone administration is adequate ventilation and not a normal level of consciousness. Opioid dependant individuals may show symptoms and signs of withdrawal after naloxone, and must be treated expectantly
- Manage pulmonary complications symptomatically
- Asymptomatic body packers: confirm bowel sounds. Administer polyethylene glycol electrolyte lavage solution (PEG-ELS) orally at a rate of 2 l/h until all packets have been passed ◈

Critical documentation

Specific agent ingested, co-ingestions and prior opioid use.

Disposition

Observe asymptomatic patients for 6–12 hours after time of ingestion. Discharge if normal mental status, normal respiratory status and naloxone not needed in last four hours.

252 Alcohol intoxication and withdrawal

Alcohol (ethanol) is used widely in many products. It is often taken in excess for recreational purposes or self-harm. Chronic ingestion leads to dependence and an acute withdrawal syndrome when intake is reduced.

The first five minutes

- ABC, VS, IV access
- Check glucose and treat if low
- Administer thiamine 100 mg IM or in IV fluids if patient appears poorly nourished
- Examine for signs of acute and subacute trauma (such as falls several days prior)

History and physical examination

Intoxication

• Common symptoms: disinhibition, dysarthria, ataxia, aggressive behaviour, flushing, tachycardia
• More serious: hypothermia, stupor, coma, respiratory depression and loss of airway reflexes. Nystagmus, ophthalmoplegia, mental confusion and ataxia could be due to thiamine deficiency (Wernicke-Korsakoff syndrome)
• Hypoglycaemia, especially in children
• Metabolic acidosis (from alcohol's effects on lactate and ketone metabolism)

Withdrawal

• Anxiety, agitation, tremor, hallucinations, hypertension, tachycardia, diaphoresis
• Withdrawal-associated seizures are generalised tonic-clonic; usually occur within 12–48 hours after the last drink (may occur after only two hours of abstinence)
• Delirium tremens – an acutely life-threatening syndrome of extreme agitation, hyperthermia, and autonomic instability; usual onset 24–96 hours after last drink

Differential diagnosis

• Consider a broad list of drugs, poisons and medical conditions. Always rule out hypoglycaemia
• Consider acute infection/sepsis (alcohol withdrawal)
• High risk for intracranial haemmorrhage (falls common, may not remember head trauma, brain atrophy may increase risk of bleed with trauma)
• Hypoxia, seizure disorder, and other metabolic disturbances (alcoholic ketoacidosis)
• Alcoholic patients often have medical complications of chronic use including hepatitis, pancreatitis, and gastritis
• Metabolic acidosis can also be caused by ingestion of toxic alcohols such as methanol, ethylene glycol, or diethylene glycol

Investigations

• Labs: electrolytes, renal (elevated anion gap, ketones), glucose, LP (if fever and altered consciousness) ◇; LFTs (elevated transaminases), ethanol (levels above 300 mg/dl associated with coma – depends on the degree of tolerance) ◈
• Imaging: CT if head injury suspected ◈

Management

The goal of acute management is to minimise the toxic effects of ingestion, or to manage the effects of withdrawal with chemical replacement.
• Protect the airway if significant risk of aspiration
• Treat hypoglycaemia
• Give IV fluids (e.g. 5% dextrose in 0.45% saline) to restore and maintain volume
• Give multiple vitamins (including thiamine) to malnourished patients
• Continue investigations as needed: do NOT attribute all AMS to alcohol, especially when patient not clearing as expected over time

Intoxication

Support ABCs, allow alcohol to be eliminated (usual rate about 25 mg/dl/hour).

Withdrawal

Benzodiazepines as needed to reduce agitation and anxiety: start with diazepam 5–10 mg IV, repeated every 5–10 minutes until symptoms are controlled (monitor RR carefully). Very large amounts may be required. Phenobarbital in 100–200 mg increments IV also useful (slow infusion with **caution** for hypotension).

Critical documentation

• Blood glucose level
• Any suspected traumatic injuries that need to be evaluated

Disposition

Admit patients who still have ALoC after several hours post ingestion. Admit patients with suspected alcohol withdrawal syndrome who do not improve rapidly with moderate doses of benzodiazepines.

253 Toxic alcohols

Ethylene glycol (antifreeze, coolants), methanol (glass cleaners) and isopropyl alcohol (disinfectants, perfumes) are grouped together as the toxic alcohols. They may be ingested by adults in self-harm attempts or as a substitute for ethanol, and are a common paediatric exposure.

The first five minutes

• ABC, VS, O_2, IV access, cardiac monitor
• Identify the source and nature of the exposure
• Co-ingested ethanol delays the onset of toxicity

History and physical examination

Ethylene glycol

• Potentially lethal ingestion > 1 g/kg
• Initial period of inebriation and sedation (similar to ethanol intoxication)
• Classically three distinct stages (can overlap considerably):
  » CNS (30 minutes–12 hours): nausea, vomiting, CNS depression, hallucinations, nystagmus, opthalmoplegia and seizures
  » Cardiorespiratory (12–24 hours): tachypnoea, hypotension, tachycardia, CCF and multi-organ failure. Most fatalities occur during this stage
  » Renal (24–72 hours): flank pain, oliguria, haematuria, proteinuria, and acute tubular necrosis

Methanol

• Potentially lethal ingestion > 1 g/kg
• Initial inebriation followed by a latent period of 12–24 hours
• Symptoms include dyspnoea, vertigo, dizziness, headache, visual disturbance (‘snowstorm’) and photophobia
• Ominous signs include seizures, coma and an afferent pupillary defect
• Patients who recover may have residual extrapyramidal movement disorders or permanent visual disturbance

Isopropyl alcohol

• The absence of early symptoms excludes a significant isolated ingestion
• CNS effects varies from inebriation and sedation, to stupor and coma
• Ketosis may be identified by a fruity breath odour (acetone accumulation)
• Large ingestions: shock, haematemesis, and pulmonary oedema

Investigations

• Labs: electrolytes, renal, ABG (methanol and ethylene glycol have a raised anion gap), serum osmolar gap (raised in all toxic alcohol ingestions) ◇; serum concentrations of the alcohols, lactate (elevated in ethylene glycol poisoning), urine oxalate crystals and urine fluorescence (poor diagnostic tests for ethylene glycol ingestion), serum acetone (elevated) and urine ketones (positive in isopropyl toxicity) ◈

• ECG ◇: (ethylene glycol can prolong the QTc interval)

Differential diagnosis

• Consider other conditions with a profound high anion gap metabolic acidosis
• Consider other conditions with a normal anion gap metabolic acidosis

Management

The goal of acute management is to increase excretion and decrease metabolism to toxic byproducts, and to maintain serum glucose and address metabolic derangement to avoid secondary injury. Absorption of ingested toxic alcohols is very rapid and they are not bound by activated charcoal.

• Supportive care: correct hypoglycaemia, ensure adequate urine output and treat seizures with IV benzodiazepines. Continuous cardiac monitor
• Haemodialysis ◈:
  » Indications include serum pH < 7.25, osmolar gap > 10, acute renal failure, visual symptoms (methanol) and deterioration despite maximal supportive care
  » End points are correction of acidosis and osmolar gap < 10

Antidotes

• Fomepizole ◈: 15 mg/kg IV bolus, then 10 mg/kg every 12 hours (ethylene glycol and methanol). It is prohibitively expensive
• Ethanol: not effective for isopropyl alcohol toxicity:
  » Indications: potentially toxic ingestion, elevated osmolar gap, high anion gap metabolic acidosis, serum pH < 7.3, serum bicarbonate < 20 mmol/l
  » Oral: loading dose 0.8–1 ml/kg of 95% ethanol or 1.5–2 ml/kg of 40% ethanol solution (e.g. brandy, gin, cane spirits) in 180 ml orange juice or water, over 30 minutes. Maintenance dose is 0.15 ml/kg/hour of > 95% ethanol or 0.3 ml/kg/hour of 40% ethanol solution
  » IV ◇: 8–10 ml/kg loading dose of 10% ethanol solution over 30 minutes to achieve a blood concentration of 1.0–1.3 g/l. Maintenance dose for an average drinker is 1.3 ml/kg/hour of 10% ethanol solution
  » Target plasma ethanol level of 1.0–1.3 g/l ◈
  » Continue until clinical signs have resolved, or until acidosis, electrolyte abnormalities and osmolar gap normalised
  » Monitor for hypoglycaemia and respiratory depression

Critical documentation

Item ingested, serial VS, response to treatment.

Disposition

Admit all symptomatic patients.

254 Toxic effects of antidepressants

The most toxic antidepressants in overdose are tricyclic antidepressants (TCAs). Selective serotonin re-uptake inhibitors (SSRIs) (such as citalopram, fluoxetine, paroxetine and sertraline), and serotonin and noradrenalin re-uptake inhibitors (SNRIs) (such as milnacipran and venlafaxine) are generally safer in overdose.

The first five minutes

• ABC, VS
• TCA ingestion may require intensive initial management: IVF, ECG, cardiac monitor, ABG, (TCA overdose > 750 mg in adults is potentially serious).

History and physical examination

Key historical features

Timing and amount of ingestion. Any co-ingestions? Intent: suicide, accidental, therapeutic.

Signs and symptoms

TCA toxicity

- *Cardiovascular*: sinus tachycardia, hypotension, conduction abnormalities, dysrhythmias
- *Anticholinergic*: flushing, dry mouth, dilated pupils, hyperpyrexia, urinary retention, constipation
- *Neurological*: drowsiness, agitation, hallucinations, hyperreflexia, myoclonus, choreoathetosis, muscle twitching or rigidity, convulsions, coma
- *Respiratory*: respiratory depression, aspiration pneumonia, adult respiratory distress syndrome, pulmonary oedema

SSRI and SNRI toxicity

Overdose with SSRIs alone rarely results in fatality and severe symptoms of toxicity only occur in large overdoses (usually more than 1 g). Fatalities involving SSRIs usually involve co-ingestion of other substances. SNRIs are mid-way in toxicity profile between SSRIs and TCAs with cardiac arrhythmias and convulsions being the most serious clinical features.

- *Gastrointestinal*: nausea, vomiting
- *Neurological*: drowsiness, tremor, serotonin syndrome (neuromuscular hyperactivity, AMS and autonomic hyperactivity), convulsions
- *Cardiovascular*: tachycardia and ECG changes (QT-prolongation)

Differential diagnosis

- Consider other causes of seizures such as isoniazid or theophylline
- Consider toxicological causes of seizures that are associated with cardiac conduction abnormalities such as antihistamines, quinine and cocaine
- Consider other causes for changes in altered mental status such as hypoglycaemia, meningitis, etc.

Investigations

TCA toxicity

- Labs: ABG (metabolic acidosis) ◇; TCA concentrations (serious cardiotoxicity > 500 g/ml (> 1 000 ng/ml can be fatal); consider serial plasma concentration monitoring as absorption may be slowed by anticholinergic effects) ◈
- ECG: ◇ (PR and QRS prolongation, ST and T-wave changes, heart block, atypical and regular VT and Vfib

SSRI and SNRI toxicity

- ECG ◇: (QT-prolongation)

Management

The goal of acute management is airway protection in altered patients, identification and treatment of respiratory depression and cardiac effects, and supportive care and monitoring while patient metabolises.

- Ensure adequate ventilation
- Treat hypotension with IVF
- Single dose activated charcoal should be considered in patients who have taken a potentially serious overdose and present within one hour of ingestion

- Cardiac dysrhythmias or wide QRS complex (> 100 msec): sodium bicarbonate ◇ 0.5–2 mEq/kg IV bolus followed by IV infusion (decreases cardio toxicity). Aim for a blood pH of 7.45–7.55. Monitor Na and K levels closely ◇; prolonged monitoring needed in potentially serious poisoning as the half-life of the TCAs vary from 24–72 hours and are increased in overdose)
- Seizures: benzodiazepines followed by phenobarbital or propofol if refractory; avoid phenytoin in TCA overdose as it may worsen cardiac dysrhythmias
- There is no role for multiple dose activated charcoal, dialysis or charcoal haemoperfusion

Critical documentation

- Record all drugs that were ingested, including the amount of the ingestion
- Rule out medical causes of AMS including hypoglycaemia

Disposition

Admit all patients with antidepressant overdose for cardiac observation regardless of the amount ingested or current symptoms.

255 Toxic effects of antipsychotic drugs

First generation neuroleptic drugs (‘typical antipsychotics’) include butyrophenones (e.g. haloperidol) and phenothiazines (e.g. chlorpromazine). Newer drugs are classified as ‘atypical antipsychotics’: clozapine, olanzapine, risperidone, ziprasidone, and others.

The first five minutes

- ABC, VS, IV, pulse-oximetry, cardiac monitor
- Glucose if altered

History and physical examination

Key historical features

- Determine the identity of the drugs taken, the quantity and time of ingestion
- Ask about co-ingestion of other drugs or alcohol

Signs and symptoms

- CNS depression is common. Seizures may occur
- Miosis often occurs due to alpha-adrenergic blockade
- Extrapyramidal dystonic reactions (torticollis, jaw muscle spasm, oculogyric crisis, rigidity, bradykinesia) and neuroleptic malignant syndrome (NMS: marked muscle rigidity, hyperthermia, lactic acidosis and rhabdomyolysis) are more common with older agents, especially butyrophenones
- Anticholinergic effects may cause tachycardia. Alpha-adrenergic blockade may cause hypotension (especially orthostatic)
- QT prolongation has been reported, but torsades is uncommon
- QRS widening with large overdoses of some older drugs (e.g. thioridazine)
- Antipsychotic drugs have been implicated in hyponatraemia (SIADH)

Differential diagnosis

- Many drugs and medical conditions can cause sedation and hypotension
- Muscle rigidity and hyperthermia may also be caused by serotonin syndrome
- Other causes of hyponatraemia

Investigations

Quantitative drug levels are not routinely available and do not help in treatment.

- Labs: glucose ◇; CK (elevated in patients with rhabdomyolysis; if CK not available, a urine dipstick positive for blood (with no red cells on microscopy) may be used as a proxy for urine myoglobin) ⟐
- Imaging: AXR (radiopaque pills (e.g. some phenothiazines) ◇

Table 255.1. Clinical effects of antipsychotic intoxication

Drug	Hypo-tension	Anti-cho-linergic	Sedation	QRS Widen-ing	QTc Prolonga-tion	Other distinctive features
Amisulpride	+++	-	+	–	+++	Bradycardia, TdP
Aripiprazole	++	+	++	–	-	Sedation
Chlorpromazine	++	+++	+++	–	+++	Extrapyramidal symptoms
Clozapine	+++	+++	+++	–	+	Agranulocytosis, sialorrhoea, seizures
Haloperidol	+	+++	+	–	+++	Extrapyramidal symptoms
Olanzapine	+++	++	++	–	+	Sedation and agitation, increases CK
Quetiapine	+++	++	+++	+	+	Tachycardia
Risperidone	++	+	+	–	+	Dystonic reactions
Ziprasidone	++	+	++	–	+++	

– = no effect, + = minimal effect, ++ = moderate effect, +++ = major effect,
CK = creatine kinase, TdP = torsades de pointes

Management

The goal of acute management is to minimise the toxic effects of ingestion, and prevent secondary injury.

- Treat hypotension with IVF, vasopressors ⟐
- Treat seizures with benzodiazepines
- Dystonic reactions: give diphenhydramine 0.5–1 mg/kg IM or IV ◇
- QRS widening: treat with sodium bicarbonate 1–2 mEq/kg IV ◇
- QT prolongation: correct underlying electrolyte imbalance (e.g. hypokalaemia or hypomagnesaemia). If torsades, IV magnesium 1–2 g IV ◇
- Hyperthermia (suspect NMS): remove clothing, spray with tepid water and place near strong fans. Consider neuromuscular paralysis
- Gastric decontamination: give activated charcoal if soon after ingestion and the airway protected. Gastric lavage NOT helpful and may increase complications

Critical documentation

All drugs taken and when, if known. Reason for ingestion (intentional self-harm or accidental). Serial VS, including temperature, and rapid bedside glucose, if altered.

Disposition

- Observe asymptomatic patients ≥ 6 hours.
- Admit patients with depressed consciousness, hypotension, or suspected NMS to ICU if available

256 Sedative hypnotics

Sedative hypnotics are a diverse group of pharmaceuticals that cause CNS depression, usually by promoting inhibitory neurotransmitter activity of GABA in the brain. Poisoning may result from suicidal overdose, unintentional misuse, or may be iatrogenic. Severe poisoning is often the result of combining sedative hypnotics with other CNS depressants such as alcohol. The most commonly used agents in this class are the benzodiazepines and barbiturates. Other agents include buspirone, chloral hydrate, gamma-hydroxybutyrate (GHB) or gamma-butyrolactone (GBL), meprobamate, and zolpidem.

The first five minutes

• ABC, IV, O_2, pulse-oximetry, cardiac monitor
• Evaluate LoC and airway reflexes. Intubate if needed
• Glucose if altered

History and physical examination

Key historical features

Time and amount of ingestion. Any co-ingestions. Intent: suicide, accidental, therapeutic.

Signs and symptoms

• Classic toxidrome: coma with normal or moderately depressed VS
• More serious: respiratory depression, aspiration, hypotension, hypothermia, cardiac dysrhythmias
• Depressed LoC may range from mild sedation, slurred speech, ataxia, and incoordination, to deep coma. Myoclonus occasionally occurs, especially with carisoprodol, methaqualone, and GHB/GBL
• Decreased RR
• Crepitations may be a sign that pulmonary aspiration has occurred
• Prolonged immobility due to coma can cause skin blisters and rhabdomyolysis

Differential diagnosis

• Always rule out hypoglycaemia in any altered patient
• Consider a broad list of drugs, poisons, and medical conditions. AEIOUTIPS is a mnemonic for Alcohol, Endocrine (and Electrolytes, Encephalopathy), Insulin (and Infection), Opiates, Uraemia, Trauma, Intracranial, Poisoning, Seizure

Investigations

Pulse oximetry ◇ is useful, but only estimates O_2 sat, not PCO_2. Serum drug concentrations may help guide decision-making with phenobarbital but are rarely available in clinically relevant time frame. Most sedative hypnotics are not detected by 'drugs of abuse' urine screens.
• Labs: ABG (may show hypercapnoea and hypoxia from hypoventilation) ◇

Management

The goal of acute management is airway protection in altered patients, identification and treatment of respiratory depression, and supportive care and monitoring while patient metabolises.
• Elevate the head of the bed, suction and reposition airway as needed to minimise risks of aspiration; intubate if needed. Monitor respiration – support if needed. Supplemental O_2 will not correct respiratory depression
• Flumazenil ⟐ is a specific benzodiazepine antagonist (dose: 0.04–2 mg IV). However, it is rarely used for acute overdose because of the risk of seizures in patients who are benzodiazepine-dependent or have co-ingested a convulsant drug
• Treat hypotension with IVF. Blankets for hypothermia
• Routine use of activated charcoal to prevent absorption or enhance elimination of drug is discouraged, especially if patient is lethargic. Gastric lavage may be considered if the patient present soon after a massive overdose, as long as the airway is protected
• Alkaline diuresis enhances elimination of phenobarbital but is of uncertain clinical value
• Consider emergency haemodialysis ⟐ in severe cases of glutethimide, meprobamate, methyprylon, or phenobarbital poisoning
• Chloral hydrate sensitises the myocardium to catecholamines and is associated with cardiac dysrhythmias. Propranolol or another beta-blocker is the drug of choice for ventricular ectopy in this setting

Critical documentation

- Specific agent, dose, and time of ingestion (if known)
- Any pre-hospital care given (e.g. decontamination measures)
- Cause and/or reason for ingestion (e.g. accidental, intentional, suicide attempt, recreational)
- Document if case is reported and where (e.g. to poison centre or other health unit)

Disposition

Admit patients with depressed level of consciousness.

Discharge patients with mild or no effects after six hours of observation.

257 Toxic effects of anticonvulsant drugs

Anticonvulsants control seizure activity by acting on one or more of the following mechanisms: sodium channel inhibition, calcium channel inhibition, inhibition of excitatory neurotransmitters, or enhanced GABA inhibitory activity. In overdose, all anticonvulsant drugs produce CNS symptoms. Some also have cardiovascular and metabolic effects (see Table 257.1).

The first five minutes

- ABC, VS, IV
- Glucose if altered
- Establish seizure precautions

History and physical examination

Key historical features

- Medication type (extended vs. immediate release), recent changes in dosing, amount and time of ingestion, possible co-ingestions
- Gather information on underlying medical history (anticonvulsants used to treat conditions such as chronic pain, migraines, mood disorders)

Signs and symptoms

Central nervous system

- Variable CNS depression is common to all anticonvulsants; signs include drowsiness, lethargy, ataxia, nystagmus, myoclonus, and coma
- Seizures can occur in overdose with carbamazepine, lamotrigine, topiramate, tiagabine, zonisamide and levatiracem
- Anticholinergic delirium can occur with carbamazepine

Cardiovascular

- Hypotension can be caused by reduced sympathetic output (e.g. phenobarbital) or direct cardiac depression (e.g. sodium channel blockade)
- Hypotension and cardiac arrest with IV phenytoin: rate and dose-related effect secondary to propylene glycol diluent

Metabolic

- Metabolic acidosis can occur with valproic acid; topiramate, zonisamide
- Electrolyte disturbances include hypernatraemia (divalproex sodium form of valproic acid), hyponatraemia (SIADH with carbamazepine), hyperchloraemia (topiramate)
- Hyperammonaemia can occur with therapeutic use or overdose of valproic acid

Gastrointestinal

- Hepatotoxicity and pancreatitis can occur with valproic acid use or overdose
- Ileus may occur due to anticholinergic effects of carbamazepine

Differential diagnosis

- AMS: many metabolic and neurologic disorders, trauma, infection, post-ictal state, and non-convulsive status epilepticus; many drugs and toxins (opioids, muscle relaxants, antipsychotics, ethanol, etc.)
- QRS widening: diphenhydramine, tricyclic antidepressants, cocaine, propranolol, venlafaxine, bupropion, antiarrhythmic drugs, hyperkalaemia, intrinsic conduction abnormalities

Investigations

Drug levels are not available for many anticonvulsants, especially newer drugs, but may aid in assessment and disposition ⟐.

- Labs: electrolytes, renal function ◇; LFTs, serum ammonia (for valproic acid) ⟐
- ECG ◇: (prolonged QRS)

Table 257.1: Common manifestations of toxicity

Drug	Common manifestations of toxicity
Carbamazepine	CNS; anticholinergic effects; hypotension; AV heart block; sodium channel blocker (similar to tricyclic antidepressants, see p. 668); hyponatraemia (SIADH)
Lamotrigine	CNS; seizures; sodium channel blocker (similar to tricyclic antidepressants, see p. 668)
Levatiracetam	CNS; seizures
Phenobarbital	CNS; hypotension; long half-life (80–120 hours)
Phenytoin	CNS; cardiodepressant effects if given rapidly intravenously (due to diluents proylene glycol)
Topiramate	CNS; seizures; hyperchloraemic metabolic acidosis
Valproic acid	CNS; elevated serum ammonia; cerebral oedema; hypotension; metabolic acidosis; hypernatraemia; hypocalcaemia
Zonisamide	CNS; metabolic acidosis; sodium channel blocker (similar to tricyclic antidepressants, see p. 668)

Management

The goal of acute management is to minimise the toxic effects of ingestion, and to prevent secondary injury due to seizure, hypoxia, and cardiac suppression.

- Most cases of overdose can be managed with adequate supportive care
- Assess airway and ventilation, intubate if needed ◇
- Volume expansion with IV crystalloids (e.g. normal saline), 10–20 ml/kg
- If vasopressor support is required, use direct-acting vasopressors such as noradrenaline ⟐ or adrenaline ◇
- For QRS prolongation >120 msec with right axis deviation and R wave >3 mm in aVR give sodium bicarbonate 50–100 mEq (1–2 mEq/kg) bolus and repeat every five minutes as needed ◇. Caution: may cause metabolic alkalosis and hypernatraemia
- Seizures: use GABA-enhancing drugs such as benzodiazepines or phenobarbital

Specific interventions

- Charcoal:
  - » Decision to administer should be individualised and used cautiously in patients with significant drowsiness who are at risk of pulmonary aspiration
  - » Carbamazepine overdose may benefit from multi-dose activated charcoal (0.5 g/kg every 4–6 hours)
  - » Consider giving charcoal in overdoses involving extended release preparations even if late presentation
- Haemodialysis ⟐:
  - » Carbamazepine: consider in severe intoxication (seizures, cardiovascular toxicity) and levels > 40 mg/l
  - » Valproic acid: refractory hypotension, seizures, renal dysfunction, severe metabolic abnormalities, and levels 700–1 000 mg/l

» Phenobarbital: deep coma, associated with hypotension and poor perfusion
- L-Carnitine: for valproic acid-induced hyperammonemia. Dose is 150–200 mg/kg/day, either orally or IV in three divided doses (maximum 3 g/day)

Disposition

- Observe asymptomatic patients for ≥ 6 hours
- Admit symptomatic patients
- Admit to ICU if cardiovascular instability, coma, or need for dialysis

258 Anti-malarial overdose

Overdose of oral agents is rare and toxic effects are similar to adverse effects observed following therapeutic doses.

Quinine

The first five minutes

ABC, VS, IV, cardiac monitor

History and physical examination

Key historical features

Symptoms at initiation of anti-malarial treatment. Duration of treatment and response to therapy. Any co-ingestions? Intent: suicide, accidental, therapeutic.

Signs and symptoms

Mild to moderate toxicity
- Cinchonism (blurred vision, vertigo, headache, tinnitus, impaired hearing)
- Skin flushing
- Nausea, vomiting

Severe toxicity
- CVS: hypotension, conduction abnormalities, ventricular dysrhythmias
- Retinal: decreased visual acuity, visual field constriction, sudden blindness, non-reactive, dilated pupils
- CNS: ataxia, coma, seizures, deafness
- Respiratory arrest

Differential diagnosis

Similar clinical picture can present with other anti-malarial agents, other type Ia antiarrhythmic agents and tricyclic antidepressants.

Investigations

Serum levels are not useful.
- Labs: CBC, electrolytes, renal, urinalysis, ABG ◇; CK ◈
- ECG: ◇ (QRS widening, QTc prolongation, ST depression, T-wave inversion, AV blocks, ventricular dysrhythmias, torsade de pointes)

Management

The goal of acute management is to mitigate the cardiotoxic effects of these drugs and to identify and treat specific side effects (e.g. hypoglycaemia, methaemoglobinaemia) promptly.
- Primarily supportive with IV fluids and antiemetics

• Consider gastric lavage ◇ and activated charcoal (single and multi-dose) ◈ for massive ingestions
• No role for haemodialysis or haemoperfusion
• QRS widening: sodium bicarbonate 1–2 mEq/kg IV bolus until QRS narrows (goal serum pH of 7.45 to 7.55)
• Torsades de pointes: magnesium 2 g, aggressive K supplementation, overdrive pacing ◇
• Unstable ventricular dysrhythmias: electrical cardioversion ◇
• Hypotension: IV ◇; adrenergic vasopressors if persistent ◈
• Retinal toxicity: usually resolves spontaneously
• Seizures: benzodiazepines, propofol or barbiturates if persistent ◇
• Hypoglycaemia: dextrose, octreotide (adult 50–100 mcg every 6 to 12 hours; child: 1 mcg/kg every 6 hours) ◈

Disposition

Admit patients with AMS, haemodynamic instability, or visual disturbances (need ophthalmology referral). Observe patients with intentional ingestion or significant ingestion (> weight/age appropriate dosing) for six hours.

Chloroquine and amodiaquine

The first five minutes

Large ingestion can be rapidly fatal (1–2 hours). Look for hypotension, hypokalaemia and QRS widening.
• ABC, VS, IV, cardiac monitor

History and physical examination

Mild to moderate toxicity
• Nausea, vomiting, abdominal pain
• Headache, neuromuscular excitability, visual and hearing disturbances
Severe toxicity
• Hypotension, any dysrhythmias
• Seizures, coma
• Respiratory arrest

Differential diagnosis

Consider medical conditions such as CNS infection, intracerebral haemorrhage, psychiatric illness, hypoglycaemia, and hypoxia.

Other toxicological causes can be sympathomimetics, other sodium channel blockers (tricyclic antidepressants) or withdrawal syndromes.

Investigations

Serum levels are not useful.
• Labs: electrolytes, renal, LP ◇; CK ◈
• ECG: ◇
• Imaging: CT brain (if intracranial causes suspected) ◈

Management

The goal of acute management is to mitigate the cardiotoxic effects of these drugs and to identify and treat specific side effects (e.g. hypoglycaemia, methaemoglobinaemia) promptly.

Mild to moderate toxicity

Symptomatic and supportive treatment.
Severe toxicity
• Treat delirium and seizures with benzodiazepines

- QRS widening or ventricular tachycardia: sodium bicarbonate (dose above)
- Hypotension: fluids, adrenaline, high dose diazepam
- Significant cardiovascular toxicity: intravenous lipids (20%) 1–2 ml/kg IV bolus, then 0.25–0.5 ml/kg/min for 30 to 60 minutes ◈
- Correct hypokalaemia cautiously
- No role for haemodialysis or haemoperfusion

Disposition

- Observe deliberate ingestions and children with unintentional ingestions for > 4 hrs; admit if symptomatic
- Admit if visual or hearing disturbances, or if symptomatic

Artemisinin derivatives (artesunate, artemether, artemotil)

- Can cause OT prolongation and neurotoxicity
- Treatment is symptomatic and supportive

Primaquine

- May cause methaemoglobinaemia or myelosuppression
- Methaemoglobineamia is treated with O_2 and methylene blue (1–2 mg/kg IV over 5 minutes with 30 ml flush of normal saline every 4 hours as needed) ◈, preferable in an ICU setting

259 Anti-tuberculosis drugs

Treatment for TB is usually divided into two phases: intensive phase where patients receive rifampicin, isoniazid, ethambutol and pyrazinamide; and a continuation phase where patients receive rifampicin and isoniazid. The drugs are usually dosed in combination tablets containing all the drugs of the treatment phase in one tablet.

The first five minutes

- ABC, VS, IV, O_2, cardiac monitor
- Establish the extent of the overdose; acute ingestion of 2 g of isoniazid (usual daily dose 300 mg) can cause neurological symptoms
- Ensure adequate ventilation in patients with neurological symptoms
- Consider in cases of benzodiazepine-resistant seizures

History and physical examination

Key historical features

Timing and amount of ingestion. Any co-ingestions? Dosage and composition of tablets (usually combination). Intent: suicide, accidental, therapeutic.

Signs and symptoms

- *Neurological*: slurred speech, dizziness, psychosis, seizures, hallucinations, coma
- *Gastrointestinal*: nausea, vomiting, diarrhoea, orange discolouration of excretions
- *Renal/metabolic*: renal impairment, INH blocks conversion of lactate to pyruvate and may be associated with severe lactic acidosis
- *Cardiovascular*: hypotension, tachycardia, ventricular dysrhythmias, cardiac arrest (severe ingestions > 15 g)
- *Ophthalmic:* optic neuropathy (ethambutol > 10 g)

Differential diagnosis

- Other causes of toxicological causes of seizures such as antidepressants or theophylline

• Other causes for changes in altered mental status such as hypoglycaemia, meningitis, stroke etc.
• Other causes of lactic acidosis and CNS symptoms (primary seizure, CNS infection with sepsis, etc.)

Investigations

• Labs: renal (glomerulonephritits with renal impairment can occur with therapeutic use), ABG (metabolic acidosis) ◇; LFT (hepatic impairment),
lactate ◈

Management

The goal of acute management is to protect the airway in altered patients, administer adequate pyridoxine to control seizures, and to manage the haemodynamic effects of acidosis.

• Mainly symptomatic and supportive
• Single dose activated charcoal should be considered in patients who have taken a potentially serious overdose and present within one hour of ingestion
• Isoniazid antidote: pyridoxine ◇
  » Indications: acute isoniazid ingestion of more than 80 mg/kg – even if asymptomatic
  » Dosing: administer dose of pyridoxine equivalent to amount of isoniazid ingested (gram for gram). Initially administer up to 5 g over one hour with remainder given over the next two hours. If ingestion unknown, administer 5 g and repeat as needed until seizures controlled. If IV preparations not available, give crushed tablets via nasogastric tube (secure the airway)
• Seizures: the primary treatment is pyridoxine ◇. Anticonvulsants that act by enhancing GABA can be used to supplement pyridoxine (IV benzodiazepines and barbiturates ◇)
• Dialysis ◈: isoniazid can be removed with dialysis, but this is usually not necessary because of the short half-life even in slow metabolisers. Rifampicin cannot be removed with dialysis

Critical documentation

Record level of consciousness and visual acuity (if ethambutol was ingested).

Disposition

Admit (to ICU if available) patients with refractory seizures, severe acidosis or haemodynamic compromise.

260 Oral antidiabetic agents

Oral antidiabetic agents are used in the treatment of non-insulin dependent diabetes mellitus. Hypoglycaemia is the main concern following overdose, which is rare following biguanide ingestion alone but may be profound, prolonged and delayed with sulphonylurea overdose. Risk factors for hypoglycaemia include the extremes of age, long drug half-life, renal impairment, malnutrition, sepsis, dehydration, drug co-ingestion and liver disease. Lactic acidosis may occur with biguanide overdose or in patients with renal impairment.

The first five minutes

• ABC, IV, neurological status, cardiac monitor
• Glucose, correct hypoglycaemia (<3.5 mmol/l (60 mg/dl))
• Do NOT give prophylactic dextrose infusions if no hypoglycaemia
• Give a single dose of activated charcoal if the time elapsed since ingestion is less than two hours. Slow-release preparations may require repeated doses

History and physical examination

Key historical features

Time and amount of ingestion. Any co-ingestions. Intent: suicide, accidental, therapeutic.

Signs and symptoms

- Onset of symptoms usually within 1–4 hours, may be delayed up to 18 hours especially if food or IV dextrose has been given
- The physiological responses to hypoglycaemia differ between different ages and genders and between patients who are treatment naïve and those on regular oral antidiabetic medications
- Early hypoglycaemic manifestations due to excess catecholamine release: palpitations, shivering, hunger, sweating, dry mouth, anxiety and confusion
- More severe hypoglycaemia causes neurological deficits such as confusion, catatonia, coma, and convulsions
- Sinus tachycardia, atrial fibrillation and ventricular premature contractions are the commonest cardiac manifestations
- Biguanides (metformin): gastrointestinal effects (nausea, vomiting, abdominal pain, diarrhoea) and lactic acidosis

Differential diagnosis

- Consider a broad list of drugs, poisons and medical conditions
- Patients on oral antidiabetic agents may have co-morbidity; consider co-ingestion of medications such as diuretics and antihypertensives

Investigations

- Labs: glucose, electrolytes, ABG (calculate anion gap if lactic acidosis is suspected) ◇

Management

The goal of acute management is detect hypoglycaemia and maintain blood glucose levels until drug cleared.

- Asymptomatic patients: allow free access to food, perform frequent glucose checks, observation of mental status
- DO NOT give prophylactic dextrose infusions
- Monitor for hypokalaemia, which can occur after administration of glucose or with correction of acidosis

Hypoglycaemia

- Hypoglycaemia is any glucose reading < 3.4 mmol/l (60 mg/dl)
- Give oral or IV dextrose if needed. Adults: 50 ml (25 g) 50% dextrose in water; children: 5 ml/kg 10% dextrose in water (📖 p. 400); neonate: 2 ml/kg (200 mg/kg) 10% dextrose in water ◇
- Recovery is usually within 5–10 minutes
- Extra boluses and continuous infusion of 5% or 10% dextrose may be required
- Monitor glucose levels frequently. If no IV access, glucagon (1 mg SC/IM; children 0.02–0.03 mg/kg) can be given although it is of uncertain benefit ◈
- Octreotide (50–100 mcg subcutaneous in adults, 1 mcg/kg in chidlren) every 12 hours for dextrose-refractory hypoglycaemia ◈
- Diazoxide (0.1–2 mg/kg/h) may be considered if dextrose infusion is ineffective and octreotide unavailable ◈
- Thiamine 100 mg IV may be given to adults with potential nutritional deprivation, e.g. alcoholism, malnutrition ◇

Metformin-associated lactic acidosis

- Sodium bicarbonate bolus and infusion for severe acidosis ◈
- Severe acidosis: haemodialysis with a bicarbonate-buffered solution ◈

Critical documentation

Document glucose level, treatment and response.

Disposition

Admit all symptomatic patients, those with documented hypoglycaemia, those with intentional ingestions and all children with a history of oral antidiabetic ingestion, particularly sulphonylureas.

Observe patients who have experienced significant hypoglycaemia for 24 hours.

261 Theophylline

Theophylline has a narrow therapeutic index and its use is becoming less common in many areas, though it is still widely available in Africa. Toxicity may occur following deliberate overdose, incorrect or inappropriate dosing, or inhibition of its metabolism by co-administered drugs such as cimetidine and ciprofloxacin. The common use of sustained-release preparations complicates management of these patients.

The first five minutes

ABC, VS, IV access, cardiac monitor.

History and physical examination

Key historical features

Prior use, including duration and dose: the spectrum of toxicity will depend on whether the ingestion is acute or chronic, with acute ingestions being more severe.

Signs and symptoms

- Gastrointestinal: nausea and vomiting (which may be severe and intractable), abdominal pain, diarrhoea, gastrointestinal haemorrhage
- Central nervous system: agitation, restlessness, dilated pupils, hyperreflexia, convulsions, coma
- Cardiovascular: tachycardia, dysrhythmias

Differential diagnosis

- B_2-adrenergic agonists (fenoterol, salbutamol, terbutaline) in toxic concentrations also cause tremor, cardiac stimulation, vomiting, hyperglycaemia and hypokalaemia
- Cocaine and amphetamine intoxication may cause cardiac and CNS stimulation
- Iron poisoning may cause similar symptoms and signs although seizures are less common

Investigations

- Labs: electrolytes (profound hypokalaemia, hyperglycaemia and metabolic acidosis) ◇; serum theophylline (therapeutic range 55–110 µmol/l (10–20 µg/ml); convulsions tend to occur at a level > 275 µmol/L (> 50 µg/ml); concentrations may continue to rise for eight hours or more after ingestion when sustained-release preparations have been ingested, requiring serial plasma theophylline monitoring) ⟐
- ECG: ◇ (cardiac dysrhythmias, including arrest)

Management

The goal of acute management is to decrease absorption, increase excretion, and control toxic effects to limit secondary injury.

- Establish IV access and replace fluids as required
- Continuous cardiac monitoring
- Give repeated or multiple dose activated charcoal (MDAC) in a patient with normal level of consciousness. MDAC may be as effective as haemoperfusion ⟐
- Control vomiting with anti-emetics such as metoclopramide. Intractable vomiting may require serotonin antagonists such as ondansetron ⟐
- Electrolytes: correct hypokalaemia
- Convulsions can be controlled by slow IV administration of diazepam

- Phenobarbital or propofol ◇ can be considered as a second-line agent. Phenytoin is not beneficial and is potentially cardio-toxic
- Dysrhythmias: beta-blocking agents (except in asthmatics), lidocaine or calcium-channel blockers (e.g. verapamil) may be useful in treating dysrhythmias ◇
- Charcoal haemoperfusion ⟐ or haemodialysis ⟐ are indicated for severe poisonings (theophylline level > 100 μg/ml (> 550 μmol/l), seizures refractory to benzodiazepines, cardiovascular compromise, patients with delayed theophylline clearance (e.g. elderly, significant liver or cardiac disease)

Critical documentation

Document whether the ingestion is acute, acute-on-chronic or chronic.

Disposition

Observe asymptomatic patients for at least 12 hours. In chronic ingestion, identify the reason for toxicity (such as drug-drug interactions, organ dysfunction or prescription error) and strategy for prevention of recurrence (patients may need baseline dose change or alternative agent).

262 Iron poisoning

Acute iron poisoning usually occurs in children who unintentionally mistake brightly coloured iron-containing multivitamins for sweets. Prenatal vitamins are a common source. Adult acute iron poisoning is usually the result of intentional overdose. Early symptoms and signs are caused by the corrosive action of iron in the gastrointestinal tract. Once absorbed, free radicals cause mitochondrial injury.

The first five minutes

- ABC, VS, O_2, IV
- Protect airway in patients at high risk of aspiration

History and physical examination

Key historical features

Timing and amount of ingestion. Any co-ingestions? Intent: suicide, accidental, therapeutic.

Calculate the amount of elemental iron ingested (toxic dose > 20 mg elemental iron/kg body weight; potentially lethal dose >180 mg/kg).

Table 262.1: Elemental iron content of ferrous salts

Ferrous salt	Amount	Elemental iron
Ferrous sulphate (dried)	200 mg	65 mg
Ferrous sulphate	300 mg	60 mg
Ferrous gluconate	300 mg	35 mg
Ferrous fumarate	200 mg	65 mg

Signs and symptoms

Acute severe iron poisoning can be divided into five phases:

- Phase 1 (0.5–6 hours): vomiting and diarrhoea (bloodstained if GIT ulceration), abdominal pain, shock, metabolic acidosis, coagulopathy
- Phase 2 (6–12 hours): symptoms improve or disappear (may be brief in severe poisoning)
- Phase 3 (12–48 hours): severe shock, vascular collapse, metabolic acidosis, hypoglycaemia, convulsions, coma
- Phase 4 (2–4 days): liver and renal failure; pulmonary oedema may occur
- Phase 5 (days–weeks): gastrointestinal scarring and obstruction in survivors

Differential diagnosis

Consider other agents that can cause vomiting with toxic ingestions such as salicylates, theophylline, organophosphates and non-steroidal anti-inflammatory drugs (see 🕮 Salicylates, p. 658, Theophylline, p. 684, Organophosphates, p. 714, NSAIDS, p. 660); other causes of GI bleed (see 🕮 Gastrointestinal bleed, p. 246 and p. 248).

Investigations

- Labs: CBC, electrolytes, ABG ◇; PT/PTT (in severe poisoning), iron concentrations (measure 4–6 hours post ingestion in significant overdoses (> 20 mg/kg elemental iron) or symptomatic patients. Repeat after 2–4 hours, but only useful within 12 hours of ingestion) ⟐
- Imaging: AXR ◇ (to diagnose significant overdose or to asses success or failure of gastrointestinal evacuation (iron is radiopaque))

Management

The goal of acute management is decreased absorption, resuscitation and control of GI bleeding.

- Activated charcoal (single or multi-dose) is not effective
- Gastric lavage ◇ can be considered if ingestion is toxic (> 20 mg/kg) and the ingestion was within one hour. There is no advantage in adding desferrioxamine, sodium bicarbonate or disodium phosphate solution to lavage fluid
- Whole-bowel irrigation ◇ can be considered if large amounts of slow-release iron tablets were ingested and a significant quantity remains in the bowel after lavage (as seen on AXR)
- Antidote: desferrioxamine ◇:
  - » *Indications*: no antidote needed if asymptomatic. Antidote needed if at least > 54 µmol/l (> 300 µgram/dl) and moderate or severe systemic toxicity (cardiovascular shock, gastrointestinal bleeding, lethargy, CNS depression, metabolic acidosis, iron-induced hepatotoxicity)
  - » *Contraindications*: severe renal disease/anuria
  - » *Dose*: continuous infusion of 15 mg/kg/hour (maximum total dose of 80 mg/kg in 24 hours). Reduce rate if blood pressure starts to drop. Continue until cessation of metabolic acidosis or clinical improvement. Generally moderate toxicity needs 12 hours of treatment and severe toxicity up to 24 hours. Prolonged therapy (> 24 hours) maybe associated with lung injury

Critical documentation

- Amount of elemental iron ingested, time of ingestion, co-ingestions
- Serial VS

Disposition

Admit symptomatic patients and those who have ingested > 20 mg/kg. Caution not to discharge symptomatic patients during in the 6–12 hour latent period as more severe symptoms may return.

263 Lithium overdose

Most cases of lithium toxicity occur with chronic therapy. Intoxication is usually precipitated by acute renal dysfunction, dehydration or concomitant medication such as thiazide diuretics or non-steroidal anti-inflammatory agents.

Usual maintenance therapeutic range is 0.6 –1.0 mmol/l.

The first five minutes

- Is lithium a new medication? (acute vs. acute-on-chronic vs. chronic toxicity)
- ABC, VS, IVF (as needed), O_2, cardiac monitor
- Stop all lithium-containing drugs (if chronic user) and any other drugs that may worsen lithium toxicity (e.g.

diuretics, ACE-I, NSAID)

History and physical examination

Key historical features

Timing and amount of ingestion. Any co-ingestions? Intent: suicide, accidental, therapeutic. Determine the type of ingestion as it influences interpretation of lithium plasma concentration:

- Acute toxicity (no previous use of lithium): symptoms may initially be absent or minor despite high serum concentrations as there is a slow distribution of lithium from plasma to the target organs where toxicity occurs
- Chronic toxicity (chronic user who develops toxicity due to increased dosing or change in renal function): symptoms occur progressively at concentrations > 1.5 mmol/l, and severity usually correlates with plasma concentrations
- Acute-on-chronic toxicity (chronic user with acute overdose): symptoms may occur more rapidly depending on the lithium level prior to the ingestion, but difficult to correlate symptoms with levels

Signs and symptoms

- CNS: changes in mental status, apathy, restlessness, tremor, hyper-reflexia, ataxia, confusion, dysarthria, convulsions, coma
- GIT: nausea vomiting, diarrhoea
- CVS: volume depletion may be the precipitating cause of toxicity or may result from lithium-induced nephrogenic diabetes insipidus (DI)
- Long-term neurological sequelae may persist for months or even be irreversible despite successful removal of lithium

Differential diagnosis

- Consider serotonin syndrome or neuroleptic malignant syndrome in patients taking psychiatric medication
- Consider other causes for changes in mental status such as hypoglycaemia, CNS infection, stroke etc.
- Consider co-ingestions of other drugs

Investigations

- Labs: CBC, electrolytes, renal (increased urea and creatinine, sodium elevated with nephrogenic DI) ◇; calcium (hypercalcaemia from lithium-induced hyperparathyroidism) ◈
- ECG changes similar to those in hypokalaemia (ST depression, T-wave inversions, prolonged PR-interval and prominent U-waves)

Management

The goal of acute management is to recognise dangerous serum levels based on ingestion type, decrease on-going absorption and increase elimination of drug.

- Consider gastric lavage in patients who ingested > 40 mg/kg less than one hour previously ◇
- Whole-bowel irrigation ◇ may be considered if lavage is not effective in significant acute ingestions
- Lithium plasma concentrations ◈:
  - » Mild toxicity: 1.5 mmol/l–2.5 mmol/l
  - » Moderate toxicity: 2.5 mmol/l and 3.5 mmol/l
  - » Severe toxicity: > 3.5 mmol/l
  - » Plasma concentrations may not always correlate with clinical severity and should take the type of ingestion into consideration (poor correlation in patients with acute or acute-on-chronic ingestion)
- IVF are often successful in eliminating lithium and should be attempted in all patients, with monitoring of urine output and serum and urine lithium and electrolytes where available
- Dialysis ◈ removes lithium from the body more rapidly and is generally indicated for severe neurological toxicity or significant renal impairment

Critical documentation

- Record type of ingestion and any co-administered prescribed medication, herbal medical or over the counter medication.

Disposition

- Admit all patients with intentional overdoses. Observe for at least 24 hours
- Refer for haemodialysis if signs of severe neurotoxicity, progressive clinical deterioration, renal failure, those who cannot tolerate fluid replacement, or lithium concentrations in excess of 4 mmol/l

264 Anticholinergic poisoning

Anticholinergic agents competitively inhibit binding of acetylcholine to muscarinic acetylcholine receptors. They include numerous pharmaceuticals (e.g. antihistamines, atropine, hyoscine, scopolamine, antipsychotics and antispasmodics), as well as plants (*Datura stramonium*, *Atropa belladonna*) and mushrooms (*Amanita muscaria*). Common antihistamines include diphenhydramine (Benadryl), chlorpheniramine, and the 'non-sedating' newer drugs, cetirizine and loratadine.

The first five minutes

- ABC, IV access, cardiac monitor
- Check for prolonged QRS
- Glucose if altered
- Check temperature and initiate rapid cooling for hyperthermia, if present
- Consider sedation for patients with agitation (see Management, below)

History and physical examination

Key historical features

Inquire about **all** possible ingestions; including herbals, plants or mushrooms. Time of onset and peak effects may be delayed because of slowed intestinal motility.

Signs and symptoms

Dilated pupils (with loss of accommodation), flushed appearance, dry skin and mucous membranes, tachycardia, urinary retention, drowsiness, agitation, delirium, coma. A useful aide memoir is:
- Dry as a bone (dry skin and mouth)
- Red as a beet (flushed skin)
- Hot as hell (hyperthermia due to agitation and impaired sweating)
- Blind as a bat (dilated pupils, loss of ability to accommodate)
- Mad as a hatter (delirium, visual hallucinations)
- Full as a flask (urinary retention)

Some antimuscarinic drugs also cause seizures due to other toxic properties (e.g. antihistamines, tricyclic antidepressants), and some antihistamines (e.g. diphenhydramine) have sodium-channel blocking effects similar to tricyclic antidepressants and can prolong QRS interval.

Differential diagnosis

- Meningitis, sepsis
- Overdose by stimulant drugs, tricyclic antidepressants
- Serotonin syndrome
- Alcohol or other drug withdrawal

Investigations

Diagnosis is clinical, and rarely, on the results of a trial of physostigmine (see below). Serum drug levels are not helpful. Other useful studies:

- Labs: electrolytes, glucose, pregnancy test ◇; paracetamol and salicylate levels, CK ◈
- ECG ◇: (wide QRS > 120 msec or tall R in aVR)

Management

The goal of acute management is to minimise the toxic effects of ingestion, and to prevent secondary injury due to agitation, seizure, hypoxia, cardiac arrhythmia or hyperthermia.

Supportive

- Treat hypotension with boluses of normal saline (10–20 ml/kg) and add a vasopressor (e.g. noradrenaline, adrenaline) if necessary ◈
- Treat hypoglycaemia with dextrose ◇
- Evaporative cooling for hyperthermia
- Benzodiazepines (diazepam 5–10 mg, lorazepam 1–2 mg) for agitation or seizures ◇
- Sodium bicarbonate (1 mEq/kg bolus IV) for prolonged QRS associated ◇
- Aggressive IVF resuscitation for rhabdomyolysis to prevent acute renal injury ◇
- Administer activated charcoal (50 gm in adults, 1 g/kg in children) for recent ingestions if the airway is intact (patient is alert) or protected ◇

Specific drugs and antidotes

- If benzodiazepines are not effective for agitated delirium, consider physostigmine (0.5–2 mg slow IV in adults, or 0.02 mg/kg max 0.5 mg in children; may repeat after 20–30 min) ◈. *Use with caution as it may cause bradycardia or heart block, especially in patients with wide QRS interval*
- Neostigmine may be useful for anticholinergic-induced ileus (0.5–2 mg slow IV in adults and 0.025–0.08 mg/kg in children) but the same caution applies with regards to risk of bradycardia or heart block ◈

Critical documentation

- Consider non-accidental injury in children
- Document the QRS interval

Disposition

Discharge patients who remain asymptomatic for six hours, and those with mild toxicity whose symptoms resolve within six hours. Admit all other patients (preferably to an ICU bed if seizures or cardiac toxicity).

265 Sympathomimetic agents

Sympathomimetic drugs mimic the effects of endogenous sympathetic neurotransmitters (catecholamine, adrenaline, noradrenalin, dopamine, etc.). Commonly found in decongestants and cough mixtures (e.g. pseudoephedrine, phenylephrine) and in appetite suppressants (e.g. phentermine, diethylpropion). Illicit sympathomimetic agents include cocaine and amphetamines.

The first five minutes

- ABC, VS, O_2, IV, cardiac monitor
- Recognition of sympathomimetic toxidrome (hypertension, tachycardia, hyperthermia, diaphoresis, minimally-reactive mydriasis, and agitation)
- Determine potential amounts ingested
- Use benzodiazepines for agitation; avoid beta-blocking agents

History and physical examination

Key historical features

Timing and details of substances used. Possible mixed substance exposure? Any co-ingestions? Intent: suicide, recreational, accidental.

Signs and symptoms

- Clinical picture varies from asymptomatic to sympathomimetic crisis (seizures, metabolic acidosis, and imminent cardiovascular collapse)
- CNS: psychomotor agitation, dilated pupils, seizures, headache, intracranial haemorrhage, focal neurologic symptoms and coma
- CVS: hypertension, tachycardia, dysrhythmias, increased myocardial O_2 demand (including ACS), and increased vascular shearing forces (aortic dissection)
- Respiratory: bronchospasm (if inhaled)
- GIT: perforated ulcers, ischaemic colitis, intestinal infarction
- Renal: urinary retention, reduced urine output
- Other: diaphoresis, hyperthermia, metabolic acidosis, rhabdomyolysis
- High mortality risk: coma, shock, body temperature > 39 °C, acute renal failure, metabolic acidosis, and hyperkalaemia
- Associated secondary complications: intracranial haemorrhage, myocardial infarction, endocarditis, aortic dissection, etc.

Investigations

- Labs: electrolytes, renal, ABG (presence and severity of metabolic acidosis) ◇; lactate, troponin, CK, LFT, urine drug tests (low utility) ◈
- Imaging: CXR ◇; echo, CT chest abdomen◈ (for complications)

Differential diagnosis

- Toxicological: theophylline, aspirin, serotonin syndrome, withdrawal syndromes, NMS
- Non-toxicological: acute psychosis, CNS infections, heat stroke, hypoglycaemia, hypoxia, thyrotoxicosis, and pheochromocytoma

Management

The goal of acute management is reduce absorption in recent ingestions, and to counter sympathomimetic effects to minimize secondary injury.

- Activated charcoal if recent ingestion and no contra-indications.
- Sedation (physical restaints very high risk):
  - » IV benzodiazepines (lorazepam 4 mg or diazepam 5–10 mg) every 10 min titrated to sedation (or midazolam 5–10 mg IM) ◇
  - » Second generation antipsychotic agents (e.g. haloperidol 10 mg IM/IV) in benzodiazepine failure ◇
- Ensure patent airway and adequate ventilation. Succinylcholine relatively contraindicated (prolonged effect, hyperkalaemia in rhabdomyolysis)
- Evaluate for rhabdomyolysis (📖 p. 617)
- Hypertensive emergencies:
  - » Benzodiazepines (dosing above)
  - » Sodium nitroprusside (0.25–0.5 mcg/kg/min) or phentolamine (2–5 mg IV) titrate to effect ◈
  - » Beta-blockers are contraindicated (unopposed α-effects)
- Hyperthermia:
  - » Antipyretics have no role; benzodiazepines (dosing above)
  - » Rapid cooling with mist spray or cold water lavage

  » Paralysis with ventilation ⟐ for severe cases ($t \geq 41$ °C)
- Treat prolonged seizures with benzodiazepines and search for other causes
- Cocaine-associated myocardial ischaemia:
  » Benzodiazepines (dosing above)
  » Aspirin 325 mg PO
  » Phentolamine 1–5 mg IV ⟐ (if SBP > 100 mmHg)
  » Beta-blockers are contraindicated (unopposed α-effects)
  » STEMI is treated according to international guidelines for non-users (📖STEMI p. 110)
- QRS widening on ECG:
  » Sodium bicarbonate, 1-2 mEq/kg IV push

Critical documentation

Serial VS, including temperature, interventions and response, serial CK levels until peaked.

Disposition

Admit patients with severe complications. Observe symptomatic patients until return to baseline level of function.

266 Cardiodepressant overdose

Many chemicals can depress cardiovascular function; they may reduce heart rate and conduction, cardiac contractility and vascular resistance.

Beta blockers (BB) act on beta-adrenergic receptors in the heart, smooth muscles, airways, and arterioles. Calcium channel blockers (CCB) inhibit the movement of calcium in the cell, thus reducing automaticity and conduction as well as cardiac contractility and smooth muscle tone. Digoxin and other cardiac glycosides have strong vagotonic effects and also inhibit the sodium-potassium ATPase pump, which affects the resting transmembrane potential and action potential of electrically excitable cells.

Table 266.1 lists the features of toxicity and specific treatments for these three groups.

The first five minutes

- ABC, VS, O_2, IV, IVF as needed, cardiac monitor
- Check glucose
- Administer activated charcoal 1g/kg orally (or via NGT) if ingestion occurred within two hours or involves a sustained-release preparation

History and physical examination

Key historical features

Determine the time of ingestion, drug ingested, and type of preparation (e.g. SR, XR). Toxic effects usually manifest with a few hours but can be delayed several hours with some extended-release drugs.
- Determine the intent of the ingestion (accidental vs. self-harm)
- Even an accidental double dose of verapamil or diltiazem can be very serious, and one pill can be enough to kill a small child

Signs and symptoms

- All cause hypotension and bradycardia (amlodipine and nifedipine initially cause reflex tachycardia because of predominant vasodilation)
- Varying degrees of atrioventricular block can be seen with all agents. Junctional rhythm is common
- Virtually any brady- or tachydysrhythmia can be seen with digoxin and other cardiac glycosides
- Nonselective beta-blockers can cause wheezing, especially in patients with bronchospastic disease
- Propranolol and other lipophilic beta-blockers can cause CNS effects including lethargy, confusion and seizures.

They also have sodium-channel blocking effects (📖 p. 668)

Differential diagnosis

- Consider a broad list of medical conditions (e.g. MI, intrinsic heart disease, hypothermia) and drugs or poisons (e.g. clonidine, tizanidine and other central muscle relaxants, opioids, cyanide, organophosphates) causing hypotension with bradycardia
- Always rule out hypoglycaemia in any altered patient

Investigations

- Labs: electrolytes (K elevated with acute digoxin poisoning), renal, glucose (elevated by CCB; depressed by BB) ◇
- ECG:◇ (repeat as needed because toxicity may worsen as the drug is absorbed and distributed; look for worsening bradycardia, increased PR interval or loss of P-wave, peaked T-waves (hyperkalaemia), AV block, wide QRS)

Drug levels are not useful for BB and CCB, but may be available for digoxin, which has a therapeutic range 0.6–2.6 ng/ml ◈.

Management

The goal of acute management is decreased absorption early in acute overdose, aggressive management of electrolyte disturbances, and supportive care for cardio-respiratory depression.

- O_2. Intubate ◇ and ventilate ◈ if needed
- Treat hypotension initially with IVF. Add vasopressors (dopamine, noradrenaline, adrenaline) if needed, although these are often ineffective ◇
- Treat symptomatic hypoglycaemia with IV dextrose ◇
- End point of therapy may include the following:
  - » Heart rate > 60 beats per minute
  - » Systolic BP > 90 mmHg
  - » Evidence of good organ perfusion (improved mentation or urine output)

Specific therapy for BB or CCB

- Glucagon (BB) ◈: 0.05–0.15 mg/kg bolus IV; may be repeated or start infusion at 0.05–0.15 mg/kg/hr
- Calcium (CCB) ◇: calcium gluconate or calcium chloride 1g slow IV push; repeat as needed to raise serum calcium level to 1.5 × normal. Caution: calcium chloride extravasation can cause soft tissue injury, use only with secure peripheral line or central line
- High dose insulin ◇ (CCB, propranolol): give regular insulin at 1 unit/kg followed by continuous infusion at 0.5–1.0 unit/kg/hr. Give supplemental glucose to maintain euglycaemia
- Lipid emulsion therapy ◈ (CCB, propranolol): 1.5 ml/kg IV, may repeat after 5–10 minutes
- Sodium bicarbonate ◇ (propranolol): dose of 2–3 mEq/kg IV if QRS interval greater than 120–140 milliseconds

Specific therapy for digoxin poisoning

- Digoxin antibodies ◈: each vial binds 0.5 mg digoxin. Estimate the number of vials needed from the dose taken or using the post-distributional serum digoxin concentration: vials = serum digoxin × body weight/100
- Atropine ◇ IV 0.5 to maximum 3 mg is often effective for bradycardia and/or AV block
- Ventricular dysrhythmias: may respond to magnesium sulphate 2 g IV; lignocaine 1–1.5 mg/kg IV followed by infusion 20–50 mcg/kg/min; or phenytoin 15 mg/kg IV ◇
- Treat hyperkalaemia with usual measures (📖 p. 426)
- Multiple dose charcoal may help eliminate digitoxin

Critical documentation

ECG: blood glucose level, treatment and response.

Disposition

Admit symptomatic patients (to ICU where available). Observe asymptomatic patients for 24 hours after ingestion of sustained-release preparations.

Table 266.1: Beta blockers, calcium channel blockers, and digoxin

Drug group	Features of toxicity	Specific treatments (see text)
Beta blockers		
All BBs	Bradycardia, hypotension, AV block, normal or low serum glucose	IVF, dopamine, glucagon
Propranolol, acebutolol	Seizures, wide QRS interval	Consider high-dose insulin, lipid emulsion
Calcium channel blockers		
All CCBs	Hypotension, bradycardia, AV block, sinus arrest, hyperglycaemia	IVF, calcium, high-dose insulin, consider lipid emulsion
Nifedipine, amlodipine	At lower doses may see hypotension with reflex tachycardia	Noradrenaline (if US shows hyperdynamic ventricle)
Cardiac glycosides		
Digoxin, digitoxin	Bradycardia, AV block, junctional tachycardia, PAT with block, hyperkalaemia	Atropine, digoxin antibodies, treat hyperkalaemia. Consider multiple dose charcoal for digitoxin

267 Toxic effects of traditional therapies

Between 60–80% of Africans use traditional medicines, and there is a common erroneous belief that they are free of side effects. Traditional medicines are usually plant-derived, with occasional animal or mineral products. Although serious adverse events are uncommon, poisoning from potent phytochemical and mineral constituents, especially cardiac glycosides and alkaloids, has been reported. Sources of toxicity include use of inherently toxic herbs; variability in potency or ingredients due to place and time of harvest, preparation or processing; and contamination or adulteration with other drugs and/or heavy metals.

History and physical examination

Key historical features

Try to identify the herbal product (with the help of botanists if possible). Ask about place of collection or purchase, purported indications, amount consumed as well as the time and duration of exposure. A picture of the product or container/label can be invaluable.

Signs and symptoms

Gastrointestinal (GI) signs and symptoms (upper GI discomfort, nausea, vomiting, haematemesis, diarrhoea and blood-stained faeces) are the most common due to the presence of GI irritants (especially cardiac glycosides) in most toxic herbs. Dysuria, haematuria and oliguria are indicative of renal toxicity. Liver injury typically presents with jaundice, hypoglycaemia, metabolic acidosis, raised liver enzymes, and an increased prothrombin time. Common CNS effects, which may be secondary to metabolic disturbances, are confusion, delirium, hallucination and convulsions.

Evaluate for toxidromes including anticholinergic, and those consistent with cardiac glycosides (digoxin-like toxicity) or heavy metal poisonings (see 🕮 Cardiac toxicity, p. 694 and Lead poisoning, p. 708). Table 267.1 outlines the organ- or systemic-specific manifestation of known herbal products.

Table 267.1: Organ and systemic toxicity in herbal poisoning

Toxic manifestation	Suspected herbs
Central nervous system	
Anticholinergic toxicity	*Atropa belladonna; Datura metel; Datura fastuosa L; Datura stramonium; Hyoscyamus niger, Mandragora officinarum*
Anxiolytic/myorelaxant/sedation	*Piper methysticum* (Kava-kava)
Altered mental status, peripheral motor and sensory neuropathy	*Averrhoa carambola, Podophyllum emodi* (mandrake) and *Podophyllum peltatum* (may apple)
Neuromuscular paralysis	*Lobelia inflata* and *Nicotiana*
Abdominal distress, metabolic acidosis, rhabdomyolysis	*Strychnos nux-vomica* (strychnine)
Dizziness, restlessness, confusion, hallucinations, respiratory failure	*Boophane disticha, Chenopodium species, Datura* species, *Scadoxus puniceus*
Seizure and coma	*Atractylis gummifera, Mitragynina speciosa*
Peripheral nervous system	
Muscle paralysis	*Anabasis sphylla, Areca catechu, Cissampelos pareira*
Cardiovascular system	
Cardiac stimulation, cardiac glycoside poisoning	*Asclepias fruticosa, Bowiea volubilis, Digitalis lanata, Ephedra* species, *Urginea sanguinea, Scilla nervosa*
Conduction blockade, bradycardia, ventricular dysrhythmias (bidirectional tachycardia), refractory cardiovascular collapse	*Aconitum* species, *Cerbera manghas, Colchicum autumnale, Taxus* species, *Thevetia peruviana, Veratrum* species
Gastrointestinal tract	
Gastrointestinal irritation, diarrhoea, vomiting	*Atractylis gummifera*, Cardiac glycoside-containing herbs, *Gloriosa superba, Jatropa curcas, Ricinus communis, Spirostachys africana*, most oral poisons
Liver	
Callilepis laureola, Colchicum autumnale, Crotalaria species, *Equisetum arvense, Heliotropium* species, *Larrea tridentate, Mentha pulegium, Piper methysticum, Senecio* species, Sho-saiko-to (a mixture of 7 herbs)	
Kidney	
Renal: interstitial renal fibrosis	*Aristolochia* species, *Equisetum arvense, Petroselinum crispum, Solidago virgaurea*
Renal potassium loss	*Glycyrrhiza glabra, Morinda citrifolia*
Renal failure	*Asparagus officinalis, Callilepis laureola, Juniperus communis, Levisticum officinale, Medicago sativa, Urtica dioica*
Haematological system	
Bleeding time, intracranial bleed	*Ginkgo biloba*
Agranulocytosis, priapism	Yohimbine
Thrombocytopenia and leucocytosis, haemolysis	*Dysosma pleianthum, Eucomis* species
Others	
Anaphylaxis, allergic reaction and contact dermatitis	Echinacea, chamomile tea, royal jelly, yohimbine, garlic and capsicum

Management

- ABC, symptomatic and cardio-respiratory supportive management remain the backbone of treatment when the specific herb consumed cannot be identified
- Activated charcoal may be considered in all patients presenting within 1–2 hours after ingestion with normal mental status and a protected airway
- Assess, monitor and correct serum electrolytes, acid-base balance, blood sugar, renal and liver functions, full blood count and coagulation status as needed. Urine should be screened for the presence of myoglobin to exclude rhabdomyolysis ◇
- Suspected cardiac glycoside ingestions require cardiac monitoring ◇. Consider digoxin antibody Fab (antigen binding fragment) ⟐ in cases with life-threatening complications (hyperkalaemia, high degree heart block, ventricular dysrhythmias, and cardiac arrest) that do not respond rapidly to conventional treatment. Infusion of 500–800 mg of Fab is adequate to treat most life-threatening ingestions in both adults and children. (Or calculated per product leaflets when serum digoxin concentration available ⟐)

Disposition

Admit unstable patients, those with serious CNS manifestations, established cardiac glycoside poisoning, or those with severe GIT symptoms.

268 Cyanide poisoning

Cyanide is one of the most potent toxins known. It inhibits mitochondrial cytochrome oxidase and leads to cellular energy failure. Exposure most commonly occurs with industrial accidents or laboratory mishaps. Cyanide is found in a variety of industries including photographic development, mining, electroplating, metal refining, and plastic manufacturing. An important source of hydrogen cyanide gas is smoke inhalation (from burning wool, silk, nitrocellulose, synthetic rubber, polyurethane). Neurotoxic effects may be seen with chronic ingestion of cyanogenic glycosides found in several plant species (*Manihot* spp, *Sorghum* spp, *Prunus* spp). A high index of suspicion is needed to make the diagnosis.

The first five minutes

- ABC, IV, 100% O_2
- IVF and vasopressors for hypotension
- Glucose if altered

Decontamination: in case of cyanide salt exposure, use appropriate PPE. Lavage with orogastric tube and instil 1 g/kg of activated charcoal (if ingestion).

History and physical examination

Key historical features

- No reliable pathognomonic symptom or toxidrome is associated with acute cyanide poisoning
- Historical clues include sudden collapse of an industrial worker, fire victim, or hobbyist who may have access to cyanide salts (e.g. jeweller, photographer)

Signs and symptoms

- Rapid onset abdominal pain, nausea, vomiting, headache, dizziness, lethargy, confusion, and syncope following exposure
- Confusion, anxiety, seizures, and/or coma may be seen
- Respiratory drive may initially increase to compensate for metabolic acidosis; respiratory depression heralds impending cardiorespiratory arrest
- Initial bradycardia and hypertension may be followed by hypotension with reflex tachycardia. Terminal features are persistent bradycardia and hypotension
- Veins and mucous membranes may appear redder than normal due to excess amounts of unused O_2 in the venous blood

Differential diagnosis

CO, hydrogen sulphide, and sodium azide also cause cellular asphyxia.

Other causes of marked lactic acidosis include sepsis, hypoxia/ischaemia, metformin intoxication, propylene glycol poisoning.

Hypoglycaemia in any patient with confusion, seizures or coma.

Investigations

- Labs: ABG (narrow arterial-venous O_2 difference is suggestive)◇; anion gap, lactate (anion gap acidosis is predominantly due to increased lactate; lactate > 8 mmol/l is associated with whole blood cyanide > 1 mcg/ml (toxic)); whole blood cyanide levels (can confirm exposure but are not available in a sufficiently rapid manner to influence initial treatment; normal range for whole blood cyanide concentration is 0.02–0.5 mcg/ml) ◈

Management

The goal of acute management removal of cyanide, restoration of aerobic metabolism, and supportive care for the

effects of lactic acidosis.

- Initiate empiric therapy with hydroxycobalamin or other cyanide antidote kit ◈
  - » Hydroxycobalamin is given as 5 g IV infusion over 15 minutes. A second dose may be given for a total dose of 10 g. Hydroxycobalamin complexes with cyanide to form cyanocobalamin (vitamin B12), which is readily excreted in urine
- If hydroxocobalamin is not available, use the conventional 'cyanide antidote kit':
  - » Sodium nitrite ($NaNO_2$) 3% IV solution over 2–4 minutes: adults – 10 ml; children – 0.33 ml/kg. Sodium nitrite induces the formation of methaemoglobin, which binds cyanide to form cyanomethaemoglobin (non-toxic compound). Caution: nitrites can cause hypotension. Do not use sodium nitrite in patients with G6PD or in fire victims with hypoxia from smoke inhalation, since methaemoglobinaemia may be dangerous in these cases
  - » Sodium thiosulphate as 25% IV solution (adults: 50 ml; children: 1.65 ml/kg body weight). This provides substrate for the enzymatic conversion of cyanide to thiocyanate (non-toxic byproduct) which is readily eliminated in urine

Critical documentation

- Nature of the exposure, i.e. suicidal, accidental, industrial etc.
- Route of exposure, i.e. inhalation, dermal, ingestion
- Pre-hospital treatment given, e.g. amyl nitrite pearls, activated charcoal

Disposition

Admit symptomatic patients (to ICU if possible). Public health officials must investigate all industrial and laboratory exposures.

269 Carbon monoxide poisoning

Carbon monoxide (CO) is a colourless, odourless, non-irritating gas produced during combustion of any carbon-containing compound (gasoline, kerosene, coal, wood, etc.). Poisoning occurs from inhalation of the gas during a fire, or with use of fuel-burning equipment such as automobiles, heaters and electricity generators in enclosed areas. Symptoms result from hypoxia as CO displaces O_2 on Hb and impairs O_2 delivery to tissues.

The first five minutes

Suspect CO poisoning in any fire victim with smoke inhalation or in persons found in an enclosed area with a potential source.

- ABC, high flow O_2 (via tight-fitting mask or ETT), IV, cardiac monitor ◇
- Treat any fire victim presenting with coma or cardiac arrest for cyanide exposure (📖 p. 700).

History and physical examination

Key historical features

- Household members with similar symptoms
- Methods of cooking, heating, lighting; any generators, automobiles, wood-fires

Signs and symptoms

- Common symptoms: headache, nausea, dizziness, confusion
- More serious: syncope, seizure, coma, cardiac arrhythmias
- Delayed neuropsychiatric deficits may occur hours to days after initial recovery
- Suspect CO poisoning when more than one patient presents with symptoms
- Mucous membranes and skin may appear red or flushed but this is not a reliable finding. Venous blood may appear more red than expected. (This might be noted as abnormally red veins on retinal exam)

- Sinus tachycardia is common. Arrhythmias, including ventricular premature beats and atrial fibrillation, may occur. Hypotension can occur with severe poisoning
- Respiratory rate often increased
- There may be varying degrees of neurological deficit depending on severity. Cerebellar findings (e.g. ataxia) have been associated with a higher risk of delayed or persistent neuropsychiatric sequelae

Differential diagnosis

Consider a broad list of drugs, poisons and medical conditions. Always rule out hypoglycaemia in any altered patient.

Drugs and toxins causing ALoC along with tachycardia and hyperpnoea (increased breathing by rate or volume) include salicylate (aspirin), metformin, methemoglobinemia, and inhalation of hydrogen sulfide or hydrogen cyanide.

Always consider concomitant cyanide poisoning (📖 p. 700) in fire victims with smoke inhalation.

Investigations

PO_2 and pulse oximetry are unreliable, giving falsely normal results.

- Labs: ABG (metabolic acidosis and hyperventilation), mixed venous PO_2 (> 60 mmHg or > 90% saturation suggests cyanide poisoning (cyanide levels rarely available)) ◇; carboxyhaemoglobin (COHb) level by co-oximetry (levels > 25% considered serious but correlation between levels and symptoms inconsistent), lactate (increased in patients with serious CO poisoning) ⟐
- Imaging: CT or MRI (damage to basal ganglia in severe cases) ⟐

Management

The goal of acute management is recognition of poisoning, restoration of oxygenation, and identification and treatment of others affected.

- O_2 ◇ in the highest available concentration (via a tight-fitting, non-rebreather mask or ETT) for at least 6–8 hours to reduce COHb levels to less than 5%
- Patients with serious toxicity (e.g. syncope, seizure, coma) must be referred to a facility with O_2
- Hyperbaric O_2 ⟐ is of uncertain benefit and is not routinely advised even in regions with access to a hyperbaric chamber
- Treat hypotension with IVF
- Smoke inhalation victims with suspected co-intoxication by cyanide may be treated with sodium thiosulfate ⟐ or hydroxocobalamin ⟐

Critical documentation

- Source of CO exposure, if known
- Neurological findings (was there a LoC? Are there any residual neurological deficits after O_2 treatment?)
- Determine whether other family members or co-workers exposed

Disposition

- Admit patients with LoC or neurological symptoms or signs
- Discharge patients with brief mild exposure, no reported LoC, with normal neuro exam – return for headache, confusion or LoC

270 Irritants and corrosives

The severity and extent of injury is directly related to the type of agent ingested; its amount, concentration, and form (solid or liquid); and the duration of mucosal contact.

Alkalis (pH > 7) cause liquefaction necrosis and penetrate more deeply (oesophagus > stomach > duodenum with ingestions). Common household products containing alkalis are oven and drain cleaners, denture cleaning agents

and automatic dishwasher detergents.

Acids (pH < 7) cause coagulation necrosis with eschar formation which may limit penetration and perforation. Upper airway injuries are common with ingestion of acids. Common household products containing acids are swimming pool and toilet cleaners, battery acid and kitchenware descaling agents.

Ingestion in children is usually accidental, and they usually expectorate most of the caustic agent before swallowing, thereby limiting injury.

The first five minutes

- Wear appropriate PPE
- Irrigate all exposure sites thoroughly with copious amounts of water
- ABC: secure early for airway oedema, stridor, IVF

History and physical examination

Key historical features

Include as many details as possible about exposure. Timing and duration on contact. Any coughing during or since ingestion? Any co-exposures? Intent: accidental, suicide. Photographs of containers/labels can be helpful.

Signs and symptoms

Caustic ingestions

- Clinical features of caustic ingestions vary widely
- Early clinical picture may not correlate with the severity and extent of injury
- Oropharynx: pain, dysphagia/odynophagia with persistent drooling, vomiting, haematemesis
- Epiglottis and larynx: hoarseness, stridor, aphonia and respiratory difficulties
- Oesophageal perforation with mediastinitis: severe and persistent retrosternal or back pain
- Oesophageal or gastric perforation with peritonitis: persistent, localised abdominal tenderness, rebound, and rigidity
- Pulmonary aspiration: dyspnoea
- Ominous signs: hypotension, tachycardia, fever, acidosis

Dermatological exposure

- Burn wounds of all degrees possible (p. 776)

Ocular exposure

- Decreased vision, photophobia, blepharospasm, red painful eye
- The eye may appear white in severe cases of alkali exposure

Investigations

Caustic ingestions

- Imaging: CXR, AXR if chest or abdominal pain ◇
- Endoscopy within 24–48 hours ◈ in patients with significant symptoms and signs. Endoscopy is contraindicated if haemodynamic instability, evidence of perforation or severe respiratory distress
- pH testing for ocular exposure

Management

The goal of acute management is decontamination to halt ongoing injury, symptomatic treatment, and infection prophylaxis in high-risk injuries.

Caustic ingestions

- Avoid the use of activated charcoal, emetics, neutralising agents, or nasogastric aspiration to remove remaining

caustic material

- Keep symptomatic patients nil by mouth, IV analgesia
- IV proton pump inhibitors should be given to prevent stress ulcers ◈
- Signs and symptoms of perforation, mediastinitis or peritonitis are surgical emergencies
- There appears to be no benefit from the use of steroids

Ocular exposure

- Irrigate the opened eye with clean water or normal saline for 30 minutes
- Topical analgesics (e.g. tetracaine drops) as needed to permit irrigation
- IV analgesics may be required
- Broad spectrum topical antibiotic (e.g. chloramphenical 0.5% drops)
- Consult ophthalmologist if pain, irritation, swelling, lacrimation persists

Inhalation exposure

- Monitor respiratory function
- O_2 and/or beta$_2$-agonist as needed

Critical documentation

- Take note of specific substance involved, time of exposure, amount and concentration of exposed substance and co-ingestions

Disposition

Admit symptomatic patients to surgery if perforation, mediastinitis or peritonitis: to ophthalmology if significant eye exposures.

271 Hydrocarbons

All petroleum distillates (e.g. paraffin, petrol) are hydrocarbons, but not all hydrocarbons are petroleum distillates (e.g. turpentine). Hydrocarbons are generally liquid at room temperature and are often mixed with agents that have systemic toxicity (e.g. heavy metals, pesticides). Hydrocarbon poisoning is relatively common (especially in children), as these are are common household products and often stored in inappropriate and unlabelled containers (e.g. kerosene for lamps, stoves). Glues, solvents, and paints are frequently used recreationally for their CNS effects by direct sniffing, 'huffing' (inhaling from a saturated cloth), or 'bagging' (inhaling from a bag over the mouth or nose).

Hydrocarbons can be classified according to their toxic potential:

- Low toxicity (high viscosity: grease, mineral and motor oils, petroleum jelly)
- Aspiration hazard is increased with less viscous hydrocarbons (paraffin, petrol, turpentine, mineral spirits, furniture polish and lighter fluids); clinical effects are typically limited to the respiratory system
- Systemic toxicity (benzene, nail polish, glues, solvents, paints, and paint removers); these hydrocarbons are readily absorbed through both the gastrointestinal and respiratory systems resulting in CNS depression and cardiac dysrhythmias

The first five minutes

- Wear appropriate PPE
- Contaminated clothing should be removed and affected skin and hair cleansed to prevent continued exposure
- ABC, VS, O_2, IV, cardiac monitor

History and physical examination

Key historical features

Timing and amount of ingestion. Any co-ingestions? Intent: suicide, accidental, recreational.

Signs and symptoms

- Fever is common at presentation. Persistent fever or a fever which develops 1–3 days post ingestion suggests secondary bacterial infection
- Respiratory: dyspnoea, tachypnoea, nasal flaring, cyanosis, wheezing and coarse crackles generally appear within 30–60 minutes after ingestion (may be delayed for 6–8 hours)
- Immediate signs of aspiration are coughing, choking, gagging, and vomiting. Chemical pneumonitis (non-productive cough, wheezing, tachycardia, fever), asphyxia, and pulmonary oedema are major pulmonary complications
- GIT: nausea, vomiting, local irritation to mucosal surfaces
- CNS: dizziness, drowsiness, headache, ataxia, blurred vision, weakness, seizures, and coma
- CVS: dysrhythmias and myocardial dysfunction

Differential diagnosis

- Other toxicological agents (iron, salicylate, carbon monoxide, paraquat, etc.) may mimic hydrocarbon aspiration
- Bronchopneumonia typically presents with fever and tachypnoea

Investigations

- Labs: ABG ◇
- Imaging: CXR (all children and symptomatic patients – four hours post ingestion; common findings include fine perihilar opacities, bibasilar infiltrates, and atelectasis) ◇

Management

The goal of acute management is supportive care, especially for pulmonary complications.

- Most patients remain asymptomatic and need no treatment
- Activated charcoal does not adsorb hydrocarbons well and should NOT be administered to patients without systemic toxicity where a single hydrocarbon was ingested. It is only indicated when adsorbable toxic additive (e.g. organophosphates) or co-ingestions are present
- Induced emesis and orogastric lavage are not recommended. However, careful nasogastric lavage ◇ may be considered for patients presenting within one hour after ingestion who have ingested a large amount of hydrocarbons with considerable potential for systemic toxicity
- Patients with pneumonitis should be treated supportively with O_2 and $beta_2$ bronchodilators. Prophylactic antibiotics and steroids are of no benefit

Secondary bacterial infections require appropriate antibiotics.

Critical documentation

- Serial VS, especially pulse oximetry
- Time of ingestion, estimated amount ingested and co-ingestions
- Suspected source and strategy for avoided further exposure
- Evaluation for risk of self-harm

Disposition

Discharge asymptomatic patients with normal CXR after six hours.

272 Lead poisoning

Lead is a heavy metal that affects many functions and organ systems in humans. Most significant exposures in adults occur in the work place (e.g. battery manufacturers, munitions, car radiators etc.), whereas environmental exposures are more important in children (leaded paint from old buildings and contaminated drinking water). Toxic lead

exposure is a reportable disease in many countries and the appropriate public health department should be informed.

The first five minutes

- ABC, VS, IV, O_2
- Control seizures and ensure adequate fluid resuscitation in patients with encephalopathy

History and physical examination

Key historical features

Symptoms are typically nonspecific. Consider lead poisoning when peculiar symptoms don't match any particular disease entity. Any illness in other household members?

Signs and symptoms

- GIT: abdominal pain ('lead colic'), constipation, anorexia, vomiting
- Neurological: headache, delirium, hallucinations, cognitive changes, behavioural abnormalities, sleep disturbances, peripheral neuropathy with extensor weakness, seizures, coma
- Musculoskeletal: joint pains, muscle aches
- Renal: nephropathy (progressive interstitial)
- Other: decreased libido, anaemia, nephropathy

Differential diagnosis

Lead poisoning mimics various medical conditions:

- Neurological: acute memory disorders, confusional states, epilepsy, GBS, mononeuropathies
- Psychiatric conditions: depression, conversion disorder, multiple chemical sensitivity
- Toxicologic: heavy metals, organic solvents
- Acute and chronic anaemias
- Gout and pseudogout

Investigations

- Labs: CBC (hypochromic microcytic anaemia and basophilic stippling of red blood cells), renal, urinalysis for protein ◇; lead level (poisoning diagnosed when ≥ 5 mcg/dl (0.24 μmol/l)), erythrocyte protoporphyrin ⟐
- Imaging: AXR (symptomatic children with an acute ingestion of lead containing objects) ◇

Management

The goal of acute management is to decrease on-going absorption, promote toxin excretion, provide supportive care and symptom relief, and control seizures.

- Activated charcoal has no benefit
- Gastric lavage or whole bowel irrigation may be used if suggestive radiopacities are observed on AXR
- Control seizures using benzodiazepines (phenobarbital is second line agent)
- Maintain an adequate urine output
- Chelating therapy ◇: blood lead levels > 45 μg/dl (2.17 μmol/l) in children and > 50 μg/dl (2.41 μmol/l) in adults

Chelating therapy

- Chelation therapy should be performed in consultation with a toxicologist or a clinician with experience using chelating agents
- Succimer (DMSA): 10 mg/kg orally every eight hours for five days, followed by 10 mg/kg twice per day for two weeks
- Calcium disodium edetate (CaNa2EDTA): 35–50 mg/kg per day as continuous infusion over 24 hours for five days (250 mg/m^2 per dose IM every four hours for five days). An additional course of $CaNa_2EDTA$ may be

given after an interruption of two days
- Dimercaprol (BAL): 4 mg/kg/dose intramuscular every 4 hours for 3–5 days
- Penicillamine: 20–30 mg/kg/day four times daily for five days, not to exceed 250–500 mg/dose

Critical documentation

Note occupational or environmental history.

Disposition

- Admit all symptomatic patients
- Household or work contacts should be screened, and removal from lead exposure (e.g. new work area) is essential

273 Methaemoglobinaemia

Methaemoglobinaemia occurs when normal ferrous Hb in red blood cells is changed to abnormal ferric haemoglobin, called methaemoglobin, which cannot carry O_2. This also increases the O_2 affinity of the remaining ferrous Hb, so it releases less O_2 to tissues. It is caused by many chemicals which oxidise haemoglobin, such as antimalarials, local anaesthetics and dapsone. It can be fatal.

Clinical cyanosis occurs when the concentration of methaemoglobin, which is dark blue, rises to about 15%. Signs of hypoxia intensify as the level rises.

The first five minutes

- Suspect methaemoglobinaemia if:
  - Cyanosis does not respond to O_2 therapy
  - PaO_2 on ABG is normal in cyanotic patient (O_2 saturation measured by conventional pulse oximetry is not reliable)
  - Blood looks chocolate-brown and remains dark when shaken in air
- ABC, highest concentration O_2 possible, IV, cardiac monitor

History and physical examination

Key historical features

- Exposure to local anaesthetics (benzocaine, lignocaine), dapsone, nitrites, aniline dyes, antimalarials, mothballs, chlorates, sulphonamides, propanil (an herbicide)
- Pre-existing anaemia, cardiac or respiratory disease may cause symptoms at lower concentrations

Signs and symptoms

- Symptoms are related to methaemoglobin concentration:
  - 10–30% – headache, lethargy, nausea, dizziness
  - 30–50% – dyspnoea, tachycardia, weakness
  - > 50% – confusion, coma, respiratory depression, convulsions, cardiac arrhythmias, acidosis and death
- Lips, tongue and mucous membranes are grey; O_2 does not change this
- Tachycardia is common. Arrhythmias may occur. Hypotension and bradycardia can occur with severe poisoning
- RR is usually increased

Differential diagnosis

- If cyanosis is due to heart or lung disease, it should respond quickly to O_2 therapy
- Consider also structural cardiac disease, especially in children, as some will not respond to O_2

Investigations

A drop of patient's blood on white filter paper is brown-red, not red. Conventional pulse oximeters are *not* helpful in methaemoglobinaemia because they give an unreliable measurement of oxygen saturation.

• Labs: ABG (may show normal PaO_2) ◇. Methaemoglobin level ◈

Management

The goal of acute management is restoration of O_2 delivery to the tissues.

- Asymptomatic patients do NOT need treatment, and usually have methaemoglobin concentrations below 20%. Cyanosis without symptoms is NOT an indication for treatment
- In symptomatic patients, give O_2 ◇ at the highest possible concentration (via a tight-fitting, non-rebreather mask or endotracheal tube)
- In symptomatic patients, give methylene blue (methylthioninium chloride) intravenously at a dose of 1–2 mg/kg (0.1–0.2 ml/kg of a 1% solution) over five minutes ◈
  - » The response is usually rapid. This dose can be repeated in one hour if symptoms persist or return. Avoid overdose (> 7 mg/kg) as this causes dyspnoea, chest pain and haemolysis

Note: patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency may get worse if given methylene blue, because it causes haemolysis. G6PD deficiency occurs in 3–26% of Africans. Severely affected patients in whom methylene blue is ineffective or not available may require exchange transfusion ◈.

Critical documentation

Identify the substance which caused the illness. Response to methylene blue.

Disposition

Admit symptomatic patients, to receive O_2 therapy and methylene blue.

274 Rodenticides

There are a variety of substances sold as 'rat poison' in different parts of Africa, and ingestion of these is a common means of suicide attempt. Accidental ingestions and intentional poisonings also occur.

Aldicarb (carbamate pesticide)

This cholinesterase inhibitor is often sold illegally as a brown or black granular substance in small plastic bags by informal street traders (see 📖 Organophosphate poisoning, p. 714).

Anticoagulant rodenticides (superwarfarins)

Warfarin and the superwarfarins inhibit the hepatic synthesis of the vitamin K-dependent coagulation factors II, VII, IX and X. The 'superwarfarins' are more potent and have a longer half-life. Rat bait contains relative low concentrations and large amounts must therefore be ingested by humans to cause serious toxicity.

Signs and symptoms

- Ingestion of large quantities (half to a full container) is associated with haemorrhagic diathesis (36–72 hours post-ingestion)
- Haemorrhagic manifestations include haemoptysis, epistaxis, gingival bleeding, gastrointestinal bleeding, haematuria, abdominal and back pain due to retroperitoneal haemorrhage, haemarthrosis, multiple ecchymotic skin lesions and even cerebrovascular accidents
- The usual cause of death is gastrointestinal bleeding

Investigations

• Labs: CBC ◇; PT/PTT (determine 48 hours post-ingestion for large or unknown quantities) ◈

Management

The goal of acute management is reversal of coagulopathy, resuscitation, and control of bleeding.

- Activated charcoal is recommended for larger or unknown quantities
- Severe cases require IV vitamin K1 at regular intervals, fresh whole blood, fresh frozen plasma and if necessary, exchange transfusions. Coagulaopathy may last for weeks to months despite treatment

Antidote: phytomenadione (vitamin K_1) ◇

- Prophylactic administration not recommended routinely
- Treat INR > 2.0
- Dose: initial adult dose in severe cases is minimum 10 mg IV (diluted in NS), at a rate ≤ 0.5 mg per minute (3–5 mg in children). Repeat INR 3-hourly; repeat or increase dose if initial response inadequate. Oral maintenance doses of 20–125 mg/day may be required in severe overdoses (usually adults) over a period of months (1–8 months). Adverse effects of IV Vitamin K: flushing, bronchospasm, tachycardia, cyanosis and cardiovascular shock
- In less severe cases, the oral dose of vitamin K_1 in adults is 15–25 mg and 5–10 mg in children
- Monitor coagulation status regularly. Morbidity or mortality is often associated with early discontinuation of therapy

Aluminium phosphide

A highly toxic fumigant pesticide usually found in tablet form. It causes widespread organ damage due to cellular hypoxia. Clinical features depend on the route of exposure. Aluminum phosphide is only available to certified agents, but occupational exposures and toxicity occur during grain fumigation in storage areas. Human consumption of three or more tablets will almost certainly result in death.

Signs and symptoms

- Inhalation: produces irritation of the mucous membranes, dizziness, numbness, nausea, vomiting, headache and diarrhoea. Severe poisonings cause acute respiratory distress syndrome, ataxia, paralysis, diplopia, congestive heart failure, convulsions and coma
- Ingestion: profuse vomiting with abdominal pain, significant hypotension, cardiac dysrhythmias, CCF, anaemia, AMS, convulsions and pulmonary oedema. Intractable shock is common. Death occurs from 30 minutes to three days post ingestion

Management

The goal of acute management is recognition of exposure, supportive care, and prevention of additional exposures to source.

275 Organophosphate poisoning

Organophosphates (OPs) are potent inhibitors of acetyl cholinesterase leading to an accumulation of acetylcholine with stimulation of muscarinic and nicotinic receptors. OPs are easily absorbed through the skin or via inhalation.

The first five minutes

- Ensure adequate provider protection (gloves, apron, mask). Decontaminate patient by removing all clothes and washing with soap and water
- ABC, VS, IVF as needed, cardiac monitor
- Ensure adequate ventilation (avoid succinylcholine – metabolised by acetyl cholinesterase, may lead to prolonged neuromuscular blockade)

History and physical examination

Key historical features

Timing, degree, and type of exposure. Any others potentially exposed?

Signs and symptoms

Symptoms and signs may appear from within minutes up to > 12 hours.

- Muscarinic effects: hypersecretion (increased sweating, salivation and bronchial secretions), constricted pupils, bradycardia and hypotension, bronchoconstriction, vomiting, diarrhoea and urinary incontinence
- Nicotinic effects: skeletal and respiratory muscular weakness, fasciculations
- CNS: restlessness, anxiety, headaches, convulsions and coma
- Intermediate syndrome can occur 1–4 days after apparent recovery (paralysis of cranial nerves, respiratory, neck and proximal limb muscles)
- A distal, sensory-motor polyneuropathy can occur 6–21 days after poisoning

Differential diagnosis

- Carbamate poisoning: similar presentation. Treat with atropine only. Avoid pralidoxime – may potentiate toxicity
- Amitraz poisoning: used as a tick ‘dip’ for cattle, sheep and dogs (see 🕮 p. 717 for similarities and differences)
- Consider CNS depressant toxicity such as opioids, clonidine, benzodiazepines, ethanol and barbiturates

Investigations

- Labs: ABG ◇; plasma cholinesterase concentration (good marker of OP exposure, but concentration does not correlate well with severity of exposure; normal range 3 000–8 500 U/L in adults, usually depressed in OP toxicity) ◈
- ECG: ◇ QTc interval or PVCs worse prognosis
- Pulmonary function: pulse oximetry, and vital capacity ◇

Management

The goal of acute management is to decrease absorption for very recent ingestions, treat muscarinic effects, reactivate acetyl cholinesterase activity, and provide supportive care including airway protection in altered patients.

- Activated charcoal if ingestion within one hour and normal level of consciousness

Antidotes

Muscarinic effects: atropine IV

- *Indication*: pulmonary symptoms (cause of death)
- *Dose*: initial IV test dose of 1 mg in adults provides a measure of severity, followed by 2–4 mg every 15 minutes until full atropinisation (control of excessive bronchial and oral secretions). Maintenance therapy: continuous IV infusion of 0.05 mg/kg/hour
- As the patient improves, reduce the dose of atropine slowly, over > 24 hours
- Observe closely as rebound effects of OP toxicity may occur. Atropine toxicity may occur at doses of > 10 mg; similar to OP CNS effects

Nicotinic effects: cholinesterase reactivator, e.g. obidoxime ◈

- Indication: consider in severe OP poisoning. Value doubtful if started = 24 hours after exposure
- Contraindication: carbamate poisoning
- Dose: 250 mg (3–5 mg/kg) IV or IM about five minutes after the first atropine dose. If good response, give 1–2 extra doses every two hours

Symptomatic and supportive

- Seizures: IV benzodiazepines ◇
- Bronchospasm: inhaled ipratropium bromide and salbutamol ◇

• Hypotension: IVF, dobutamine ⟐

Critical documentation

• Source of OP exposure if known, other patients involved

Disposition

Admit all patients (observe for respiratory distress). Rebound effects of OP toxicity may occur.

276 Other pesticides/herbicides

There are several pesticide/herbicide agents whose toxicity may mimic some symptoms of OP poisoning. It is crucial to identify these exposures accurately, as the treatments for OP poisoning may exacerbate the toxicity of these agents.

Respiratory arrest is the leading cause of death.

The first five minutes

• Ensure provider protection (gloves, apron, mask). Decontaminate patient: remove all clothes and wash with soap and water
• ABC, VS, IVF and O_2 as needed (avoid O_2 if paraquat suspected), cardiac monitor

History and physical examination

Key historical features (all agents)

Timing, degree, and type of exposure (e.g. ingestion, skin absorption, inhalation). Possible mixed substance exposure? Any others potentially exposed?

Amitraz

Amitraz is used as a veterinary ectoparasiticide (tick dip) and agricultural insecticide. It acts as a α_2-adrenoceptor agonist.

Signs and symptoms

• Rapid neurological depression (drowsiness, coma)
• Respiratory depression/arrest
• Miosis
• Bradycardia, hypotension
• Hypothermia
• Hyperglycaemia

Differential diagnosis

• Amitraz toxicity is frequently misdiagnosed as OP poisoning
• Consider other substances (carbamate, opioid, clonidine)

Management

The goal of acute management is to correctly identify the exposure (and avoid harmful pralidoxime therapy for misdiagnosed OP poisoning), to decrease absorption for very recent ingestions and to provide supportive care including airway protection and ventilatory support.
• Activated charcoal if ingestion within one hour and normal level of consciousness
• Intubation ◇ and ventilation ⟐ as needed

Table 276.1: Comparison of clinical presentation between amitraz and cholinesterase inhibitors

	Amitraz	**Organophosphates/carbamates**
Similarities	Impaired consciousness, respiratory depression, bradycardia, miosis	
Differences	Dry mouth Hypothermia Decreased gastrointestinal motility	Excessive salivation Excessive sweating Urinary and faecal incontinence Muscle fasciculation

Chlorinated hydrocarbon insecticides (organochlorines)

Endosulfan, gamma-BHC, chlordane, endrin, DDT and dieldrin.

Signs and symptoms

- Mild poisoning: nausea, vomiting, dizziness, paraesthesias, tremors
- Significant poisoning: seizures, confusion, ataxia, myocardial irritability, respiratory depression, metabolic acidosis and impaired liver function

Management

The goal of acute management is to correctly identify the exposure (and avoid potentially harmful atropine therapy for misdiagnosed OP), to decrease absorption for very recent ingestions, and to provide supportive care including airway protection in altered patients.

- Activated charcoal if ingestion within one hour and normal LoC
- Intubation ◇ and ventilation ◈ as needed

Glyphosate

Glyphosate herbicides are widely use in agriculture. Although exposures are common, severe toxicity is rare.

Signs and symptoms

The amount ingested correlates with systemic manifestations.

- Mild poisoning: nausea, vomiting, abdominal pain, mouth and throat irritation/ulcerations
- Significant poisoning: severe mouth, throat or gastrointestinal mucosal ulceration, metabolic acidosis, renal and hepatic impairment, pulmonary oedema and respiratory distress
- Skin burns (rarely severe)
- Mild conjunctivitis with eye exposure

Differential diagnosis

- Caustic substances

Management

The goal of acute management is symptomatic and supportive treatment. Decontamination is not indicated.

Paraquat and diquat herbicides

Paraquat is the most toxic herbicide and any oral exposure should be considered potentially fatal. Paraquat is poorly absorbed through intact skin but exposure to the eyes can cause corneal damage. Moderate inhalation of spray is unlikely to cause serious systemic toxicity.

Signs and symptoms

- Oral, oesophageal and gastric erosions with severe gastro-enteritis
- Abdominal pain, nausea, vomiting, diarrhoea, dysphagia and coughing

- Renal and liver failure develops within 1–3 days
- Pulmonary oedema, ARDS and pulmonary fibrosis
- Large doses cause death within 24 hours due to metabolic acidosis, pulmonary oedema, myocardial depression and coma

Investigations

- Labs: ABG, electrolytes and renal function, liver function ◇
- CXR ◇

Management

The goal of acute management is to decrease absorption, increase excretion, and provide supportive care.

- Avoid O_2 (it increases paraquat's toxicity)
- Activated charcoal and/or gastric lavage are indicated
- Appropriate supportive care
- Haemoperfusion ⟐

Pyrethroid insecticides

The pyrethroids are the most widely used insecticides in the household setting (available as sprays, foggers and mosquito coils), and are generally non-toxic in doses commonly ingested.

Signs and symptoms

- Allergic and hypersensitivity reactions
- Nausea, vomiting and tremors

Management

The goal of acute management is symptomatic and supportive. Administer activate charcoal if no contra-indication.

277 Toxic plants

Exposure most frequently involves children (> 90% with no or minimal effects). Serious toxicity occurs with large ingestions of highly toxic species.

The first five minutes

- ABC, VS, IV, cardiac monitor as needed
- Blood glucose

History and physical examination

Key historical features

Try to identify the plant. Ask about the place found, amount consumed, as well as the time and duration of exposure. A picture of the plant can be invaluable. Ask about other household members with symptoms.

Signs and symptoms

Check for hypoglycaemia in all patients.

- CNS: seizures, visual hallucinations, coma
- Gastrointestinal:
  - Mucosal erythema and mild swelling
  - Mild gastroenteritis (self-limited): nausea, vomiting, diarrhoea

» Severe gastroenteritis with systemic toxicity: GI bleeding, shock
- Cardiac:
  » Cardiac glycosides (digitalis poisoning): vomiting, diarrhoea, confusion
  » Alters sodium-channel activity: bradycardia, tachycardia, VT, VFib
- Cholinergic features:
  » Primary nicotinic (muscle fasciculations, seizures, weakness/paralysis, tachycardia, hypertension, coma)
  » Secondary muscarinic symptoms (diaphoresis, miosis, lacrimation, salivation, wheezing, bradycardia, vomiting, diarrhoea)
- Anticholinergic features:
  » Delirium, hallucinations, seizures, mydriasis, tachycardia, decreased bowel sounds, urinary retention, flushed, dry and warm skin
- Cyanide features:
  » Cherry-red skin, severe tachypnoea, tachycardia and hypotension

Differential diagnosis

- Acute gastroenteritis (viral/bacterial)
- Consider other toxicological and medical causes of seizures, cardiac conduction abnormalities, AMS

Investigations

Consider according to clinical picture.
- Labs: CBC (Low Hgb), electrolytes (hyperkalaemia), renal (renal impairment), glucose, ABG (metabolic acidosis) ◇; CK, urine myoglobin◈
- ECG: ◇

Management

The goal of acute management is early identification of the causal agent and supportive care.
- Ensure patent airway and adequate ventilation; IVF replacement
- Local irrigation (for skin or eye exposure)
- Activated charcoal for alert patient, within one hour of highly toxic plant ingestion
- Anti-emetics (ondansetron 0.15 mg/kg IV, max 4 mg) ◈
- Treat seizures with benzodiazepines (lorazepam 0.05–1 mg/kg, max single dose 2–4 mg), phenobarbital (15–20 mg/kg, may repeat 5–10 mg/kg every 15–20 minutes as needed, max total dose 40 mg/kg) as second line ◇
- If hallucinating give benzodiazepines, quiet and dark room
- Cold milk, popsicle or ice cream for oral mucosal irritation

Specific treatment

Cardiac
- Glycoside induced dysrhythmias: digoxin-specific Fab fragments 400 mg (10 vials) IV over 20 minutes, then 400–800 mg IV over 4–8 hours ◈
- Clinically significant bradycardia (non cardiac glycosides): atropine (0.02 mg/kg IV, min 0.1 mg, max 1 mg); adrenaline (0.01 mg/kg, max 1 mg, slow infusion) and/or cardiac pacing ◈ if unresponsive
- Ventricular arrhythmias: amiodarone (5 mg/kg IV) ◇
- Widened QRS: sodium bicarbonate (1 mEq/kg IV bolus, max dose 50 mEq) ◇
- Torsades de pointes: magnesium sulphate (50 mg/kg IV, max dose 2 g ◇

Cholinergic poisoning (see Organophosphates, p. 714)

- Initial IV test dose of 1 mg atropine in adults provides a measure of severity, followed by 2–4 mg every 15 minutes until full atropinisation. Maintenance therapy: continuous IV infusion of 0.05 mg/kg/hour

Anticholinergic poisoning (see Anticholinergics, p. 690)

• Physostigmine (0.01–0.03 mg/kg slowly over 5 min, max dose 2 mg) ◈
• Benzodiazepines if agitated

Cyanide poisoning

• Hydroxocobalamin (70 mg/kg IV, max 5 g) with sodium thiosulfate 25% IV (1.65 ml/kg / 412.5 mg/kg) ◈

Critical documentation

Clinical findings and identification of the ingested plant are essential for determining specific management.

Disposition

Admit symptomatic patients (other than mucosal irritation). Observe asymptomatic patients until six hours post-ingestion.

Table 277.1 High toxicity plants

Common name	Scientific name	Clinical picture
Autumn crocus/meadow saffron/naked lady	*Colchicum autumnale*	Gastro-enteritis with systemic toxicity
Azalea	*Azalea* (multiple species)	Cardiac arrhythmias, AMS
Baby woodrose/elephant creeper/woolly morning glory	*Argyreia nervosa*	Hallucinations
Castor (oil) bean	*Ricinus communis*	Gastro-enteritis with systemic toxicity
Chewed pits or seeds from cherry, apricot, peach, plum, pear, apple	*Prunus* (multiple species)	Cyanide toxicity
Deadly nightshade	*Atropa belladonna*	Anticholinergic poisoning
Foxglove	*Digitalis purpurea*	Cardiac arrhythmias
Henbane/stinking nightshade/black henbane	*Hyoscyamus niger*	Anticholinergic poisoning
Jimson weed/datura	*Datura stramonium*	Anticholinergic poisoning
Monkshood/aconite/women's bane/devil's helmet/blue rocket	*Aconitum spp*	Cardiac dysrhythmias, gastro-enteritis, AMS
Morning glory	*Ipomea violacea*	Hallucinations
Oleander	*Nerium oleander*	Cardiac arrhythmias
Poison hemlock/poison parsley	*Conium maculate*	Nicotinic poisoning
Pokeweed	*Phytolacca americana*	Gastro-enteritis
Prayer/Jequirity bean	*Abrus precatorius*	Gastro-enteritis with systemic toxicity
Rhododendron	*Rhododendron*	Cardiac arrhythmias, AMS
Water hemlock	*Cicuta maculate*	Status epilepticus
Wild tobacco	*Nicotiana tabacum*	Nicotinic poisoning
Yew trees	*Taxus* spp	Cardiac arrhythmias

278 Seafood toxins

Illness after eating seafood may be due to toxins, contamination by infectious organisms or allergy; this chapter reviews toxin ingestions only.

The first five minutes

Muscle weakness caused by paralytic shellfish poisoning or tetrodotoxin poisoning can lead rapidly to respiratory failure, so measures to assist ventilation should be readily available.
• ABC, IVF, cardiac monitor ◇
• Activated charcoal may be beneficial if given within one hour of ingestion, unless there are already significant gastrointestinal symptoms

History and physical examination

Key historical features

There is usually a history of ingestion of seafood within minutes to 24 hours of presentation. Others exposed or ill?

Possible causes and differential diagnosis

See Table 278.1.

Investigations

Guided by clinical presentation. There are no available specific toxin tests.

Management

- The goal of acute management is symptomatic management
- IVF, O_2, assisted ventilation as required; inhaled beta-agonists for bronchospasm
- Antihistamines and H2-receptor antagonists (e.g. cimetidine) for scombroid poisoning
- Mannitol infusion is no longer recommended for ciguatera poisoning

Critical documentation

- Presence or absence of neurological features
- Alcohol co-ingestion, as may increase the severity of ciguatera symptoms
- Source of exposure, for public health notification

Signs and symptoms

Table 278.1 Causes, differential and signs and symptoms of seafood poisoning

Type of poisoning	Toxins	Clinical presentation
Scombroid	Ingestion of improperly stored red-muscled fish (e.g. kingfish, wahoo, mackerel, tuna, skipjack, marlin, pink salmon, anchovies, herring, pilchards, sardines), rich in histadine, which is converted to histamine; may have a peppery or metallic taste	Histamine toxicity (flushing, skin rash, urticaria, sweating, headache, nausea, vomiting, diarrhoea, anxiety, palpitations). Occasionally, dyspnoea, bronchospasm and hypotension.
Tetrodotoxin	Ingestion of tetrodotoxin-rich organs (liver, ovaries, intestines, skin) of mainly puffer fish. Tetrodotoxin is heat-stable, therefore not destroyed by cooking.	Neurological (perioral and peripheral paraesthesiae, motor paralysis, salivation, twitching, ataxia, convulsions, respiratory failure) and gastrointestinal (nausea, vomiting, abdominal pain). Occasionally, cardiac arrhythmias.
Shellfish	A proliferation of dinoflagellates in the sea causes a visible 'red tide'. Shellfish concentrate dinoflagellate toxins, which cause clinical poisoning when ingested. Toxins can be inhaled when aerosolised by wave action. Toxins are mostly heat stable.	Paralytic shellfish poisoning: neurological with paraesthesiae, weakness, ataxia, cranial nerve dysfunction resulting in dysarthria, dysphagia and transient blindness; some gastrointestinal features. Neurotoxic shellfish poisoning: parasympathetic and adrenergic stimulation resulting in paraesthesiae, hot/cold dysaesthesia, ataxia, muscle twitching, bradycardia, dilated pupils, NO paralysis; aerosolised toxins cause respiratory irritation. Amnesic shellfish poisoning: gastrointestinal followed by neurological effects such as memory loss, focal deficits, seizures. Diarrhoeal shellfish poisoning.
Ciguatera	Ingestion of ciguatoxin-containing reef fish, e.g. parrotfish, surgeonfish, barracuda, moray eels, amberjacks, grouper, reef sharks, wrasses, scavengers, snappers. Ciguatoxin is heat-stable, therefore unchanged by cooking or refrigeration methods.	Gastrointestinal (nausea, vomiting, abdominal pain) followed by neurological (perioral and peripheral paraesthesiae, hot-cold dysaesthesia, ataxia). Other features include myalgia, arthralgia, headache, pruritis, sweating, hiccoughing, dysuria, bradycardia, hypotension.

Disposition

Admit patients who ingested tetrodotoxin-containing fish or have eaten shellfish from the same source as someone who has developed paralysis. Observe symptomatic patients until stable. Discharge asymptomatic patients.

Patients with ciguatera poisoning should avoid alcohol and fish in the recovery phase (paraesthesiae, pruritis and fatigue may persist for months or even years. Can use amitriptyline for pain and antihistamines for pruritis).

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Part

4

S. Trauma

279 Advanced trauma life support and principles of trauma management

The management of the severely injured patient requires a well-defined system of care. The system below is adapted from the American College of Surgeons' Advanced Trauma Life Support (ATLS) course.

Preparation

Ideally, team is notified prior to arrival of the trauma patient and equipment and personnel prepared.

Patients with any of below are at high risk:

Mechanism: high speed collision, any pedestrian vs. motor vehicle, fall from over 3 m
Injuries: serious penetrating injuries, evisceration, explosion, major limb amputation, severe burns
Signs: RR > 30 per minute, SBP < 100 mmHg (adult), GCS < 14

Treatment: assisted ventilation, chest decompression

The ideal trauma team:

Medical trauma team leader (emergency physician or trauma surgeon)
Dedicated airway doctor
Procedure doctor (trainee emergency physician or surgeon)
Trauma nurse leader (scribe)
Airway nurse or respiratory technician
Circulation nurse
Radiographer

Primary survey

Includes initial assessment and management of all immediately life-threatening injuries, in order of priority. Once identified, life-threatening issue(s) must be dealt with before proceeding. If the patient's condition deteriorates, the primary survey must be repeated.

Primary survey:

A = Airway maintenance with cervical spine immobilisation
B = Breathing
C = Circulation with haemorrhage control
D = Disability: neurologic status
E = Exposure

A = Airway

If the patient is talking normally, the airway is patent. Abnormal voice requires further evaluation. Signs of airway obstruction include:

- Agitation, hypoxia, obtundation, cyanosis, accessory muscle use or visible obstruction (e.g. vomit or blood in the mouth)
- Abnormal airway sounds, including stridor and gurgling

Immediate airway management may include:

- Jaw thrust
- Suction
- Oral or nasal airway placement
- Endotracheal intubation

Indications include:

- Threatened airway (airway burns, bilateral mandibular fractures, laryngeal oedema, tracheal injury, expanding neck haematoma)
- Respiratory failure
- Inability to protect a patent airway (patients with GCS < 9, with airway haemorrhage, or requiring sedation

While managing the airway, assume that the cervical spine has an unstable injury requiring manual in-line immobilisation (replaced by well-fitting hard collar, or sandbags/blanket roll secured with two tapes at the earliest opportunity).

B = Breathing

Administer high flow O_2 to keep sats > 93%, and manage immediately life-threatening injuries:

Tension pneumothorax

- *Assessment:* respiratory distress, unilateral decreased breath sounds, hypoxia, tachycardia, hypotension, cyanosis, tracheal deviation towards unaffected side
- *Management:* immediate needle decompression (2nd intercostal space, mid-clavicular line) and placement of chest tube). (📖 p. 838)

Open pneumothorax

- *Assessment:* respiratory distress, hypoxia, tachycardia, cyanosis, open ‘sucking’ chest wound
- *Management:* placement of occlusive ‘one-way valve’ dressing over open wound – dressing secured only on three sides and not adherent to chest wall (allowing outflow of air from pleural space, but not inflow), followed by placement of chest tube. (📖 Procedures p. 838)

Flail chest

- *Assessment:* respiratory distress, hypoxia, tachycardia, cyanosis, flail segment (localised portion of chest wall moves in opposite direction during spontaneous respiration)
- *Management:* consider early positive pressure ventilation (in addition to administering high-flow O_2). Place chest tube prior to PPV if any concern for PTX

C = Circulation

The key circulation priorities are to identify shock, determine its cause and fix the problem. By far the most common cause is haemorrhage. Clinical features may include tachycardia, hypotension, sluggish capillary refill, AMS, and other signs specific to the source of haemorrhage. The absence of hypotension does *not* exclude shock as hypotension is a late finding.

Potential sources of shock:

Chest: massive haemothorax, cardiac tamponade, tension pneumothorax
Abdomen: hollow or solid organ injury with haemorrhage
Pelvis: fracture with vascular disruption
Long bones: fracture with haemorrhage ± associated vascular injury
Peripheral vascular injury or other source of external haemorrhage
Spinal trauma with neurogenic shock

See individual chapters for specific clinical findings, indications for emergency surgery and other management.

In parallel with steps to stem the bleeding, administer high flow O_2, place two large-bore IV lines, and bolus 20 ml/kg crystalloid with frequent reassessment during bolus (patients without evidence of shock can be given smaller aliquots). The persistence of shock after two crystalloid boluses mandates the rapid transfusion of blood and clotting factors to prevent coagulopathy.

D = Disability

Rapidly assess for evidence of head or spinal injury; record rapid blood glucose, GCS, pupil size and reactivity, basic neurological examination (extremity movements). Consider neurogenic shock in patients with persistent hypotension despite resuscitation.

E = Exposure

By the end of the primary survey, the exposed patient should be log-rolled to allow complete inspection. Maintain normothermia.

Investigations

In parallel with primary survey: CXR ◇, pelvic XR ◇, focused assessment with sonography (FAST) ◈. Order additional XR and CT ◈ as indicated.

Note that the cervical spine cannot be cleared by lateral C-spine XR in the polytrauma patient or patient with altered consciousness. Maintain C-spine immobilisation until definitive clearance.

Secondary survey

Following the primary survey, conduct a detailed head-to-toe examination documenting all other injuries. This is also an opportunity to take the injured patient's history, including allergies, medications, past history, last meal and mechanism, (AMPLE history). Place a urinary catheter, if indicated.

280 Approach to the paediatric trauma patient

Children decompensate later than adults, but deteriorate more rapidly. They also exhibit different injury patterns to adults. Children have 'resilient bones': serious internal organ injuries may occur without overlying skull or rib fractures. Common management problems include over- or under-resuscitation, medication errors, and failure to recognise hypothermia and hypoglycemia.

Always consider the possibility of non-accidental injury if:

- Inappropriate delay to presentation
- Mechanism of injury incompatible with developmental level
- Inconsistent history
- Specific pattern of injury suspicious for child abuse (📖 p. 906).

Resuscitation and primary survey

Follow the ABCDE approach (see 📖 ATLS, p. 726). Use a Broselow tape or similar to guide management (📖 Paediatric resuscitation, p. 18).

A = airway and c-spine protection

Indications for cervical spine protection:

- High speed MVA
- Fall from height (> twice child's height)
- Polytrauma
- Altered mental status
- Coma, neurological deficit
- Tenderness over the cervical spine
- Distracting injuries.

If the patient is talking normally, the airway is patent. Abnormal voice requires further evaluation. Interventions include:

- Jaw thrust, suction, foreign body removal, oral or nasal airway placement
- Indications for intubation ◇ include (📖 p. 18):
  - » Threatened airway (airway burns, bilateral mandibular fractures, laryngeal oedema, tracheal injury, expanding neck haematoma)
  - » Respiratory failure
  - » Inability to protect a patent airway (patients with GCS < 9, with airway haemorrhage, and those requiring sedation)

Use rapid sequence intubation ◇:

- Suxamethonium (1–1.5 mg/kg IV) PLUS etomidate (0.2–0.4 mg/kg IV) or ketamine (1–2 mg/kg IV)

- ETT size: (age/4) + 4

B = breathing

Examine for life-threatening chest injury – management as for adults (see Advanced trauma life support, p. 726).

C = circulation

- Assess vital signs (VS) and peripheral perfusion for signs of shock
- Normal systolic BP for a child is > 70 + (age × 2) (See p. 928)
- IV access: no more than 3 attempts lasting < 90 seconds. If unsuccessful: IO access (see Intraosseous infusion, p. xx)
- Fluid bolus: 10–20 ml/kg crystalloid. Reassess after infusion. Persistent shock: repeat. If still shocked after 2 fluid boluses, give 5–10 ml/kg blood (packed cells). Consider fresh frozen plasma (FFP) and platelets if transfusing > 30 ml/kg packed cells. Repeat evaluation for ongoing bleeding and transfer to surgery

D = disability

- Assess consciousness with age-appropriate GCS (see p. 937)
- Intubation if GCS < 9

E = exposure and temperature control

- Avoid hypothermia: children cool quickly. Warm infusion fluids, cover with warm blanket
- Cut away clothes – avoid spinal manipulation

Take an AMPLE history and perform a secondary survey

- Example: allergies, medication, past medical and surgical history, last meal and events leading to the injury
- Secondary survey is aimed at identifying all injuries. It should only be performed once the ABG are stabilised and should include a log-roll

Imaging

- A lateral c-spine (see Neurological imaging, p. 734) and CXR ◇ are required if XR facilities are available. All other XRs as indicated
- CT ◈:
  - » Head injuries: see Neurological imaging, p. 734 for indications
  - » Multi-trauma patients: CT of entire torso
- FAST ◈ useful in chest and abdominal trauma. Positive – referral for possible surgery

281 Trauma in the pregnant patient

Any female trauma patient aged 10–50 should undergo pregnancy testing. Pregnancy causes many changes in maternal physiology and, beyond the age of fetal viability, adds considerations of fetal well-being. Common mechanisms are falls and MVC, and even seemingly minor trauma may result in substantial maternal or fetal morbidity. Women suffering trauma in the third trimester are at risk for abruptio placentae, uterine rupture, and premature labour. Remember: resuscitation of the mother resuscitates the foetus.

Placenta and fetal circulation have substantial impact on maternal circulation. In gestation > 24 wks, C-section may improve maternal haemodynamics (all perimortem patients) or fetal distress (with viable fetus).

The first five minutes

- ABC, IV access, O_2 facemask (see ATLS p. 726)
- If > 20 wks gestation, left lateral positioning (15–30° on spine board) to prevent compression of IVC by gravid uterus.

- Primary survey:
  - A: early intubation with NGT placement
  - B: SIMV mode when ventilation required; caution for high diaphragm if chest tube placed
  - C: check for vaginal bleeding
  - Disability – always consider eclampsia if seizures
  - Exposure – keep normothermic
- C-section may be appropriate in the setting of: imminent maternal death, CPR ineffective within four minutes, or distress in fetus of viable age (≥ 24 weeks, depends on available resources for neonatal resuscitation)

History and physical examination

Key historical features

- Gestational age, Rh status, and any pregnancy complications. Mechanism of injury.

Signs and symptoms

Anatomical and physiological changes of pregnancy:

- Hyperlordosis with resultant change in centre of gravity
- Pubis: softening in third trimester
- Diaphragm: high riding
- Bowel: slower gastric emptying
- Increased respiratory rate, tidal volume, lower functional residual volume
- Respiratory alkalosis usual, so 'normal' pH = acidosis
- Lower SBP and DBP, with relative hypervolaemia and haemodilution anaemia
- More than 25% flow reduction leads to insufficient fetal perfusion

Critical signs and symptoms:

- Maternal perfusion and fetal HR
- Fetal monitoring (CTG) recommended in all viable pregnancies ⟐ and late decelerations indicate fetal distress (esp. placental abruption)
- Consider hand-held fetal Doppler heart monitoring ◇

Common conditions in pregnant trauma patients

- Preterm labour ± premature rupture of membranes:
  - High incidence – due to blood stimulation of the myometrium
- Abruptio placentae ± amniotic fluid embolism:
  - Uterine tenderness, spasm and > 8 contractions/hour
- Uterine rupture
- Penetrating trauma
- Seizures: CT head and screen for eclampsia

Investigations

- DO perform all necessary imaging – when possible, shield uterus and use non-ionising method. US whenever possible
- Evaluate for Rh incompatibility in all trimesters; Kleihauer-Betke test for Rh negative mother > 12 wk gestation

Management

The goal of acute management is optimisation of maternal and then fetal wellbeing.

- Basic resuscitation, left lateral positioning
- Six hours of fetal monitoring recommended for all > 20 wk gestation ◇
- Consider early Rh-antibody (300 µg IV/IM, CHECK local formulation as some IM only) for Rh-negative mother
- Consider early steroid therapy for fetal lung maturation if > 20 wk

- Preterm labour: exclude abruptio and then consider suppression of labour:
  - Beta-stimulants, Ca-antagonists and NSAIDs have been used
  - Atosiban is a newer (expensive) tocolytic agent
- Arrange appropriate transfer to OT or other facility. Unstable patients need emergency laparotomy!
- Emergency caesarean for viable fetus; however allow natural vaginal delivery for non-viable fetus

Critical documentation

Fetal movements and heart sounds, or CTG; any surgical intervention

Disposition

- Transfer from limited facilities ASAP after head-to-toe exam
- Receiving facility should have surgical and obstetric capacity

282 Approach to neuro-imaging: trauma

This is a brief guide to the indications for neuro-imaging in trauma. CT is usually the study of choice for head trauma, given its diagnostic test performance and rapidity, although MRI is sometimes recommended to reduce radiation exposure in stable children. The relative utility of skull X-ray (SXR) is limited by significant radiation exposure and poor diagnostic test performance.
See (📖 Skull x-ray, p. 952) for discussion. Patients with head trauma who do not undergo imaging should be observed for at least 24 hours.

Indications for CT Head

Adults

- Glasgow Coma Score (GCS) < 13 (📖 GCS, p. 937)
- GCS of < 15 at two hours post-injury
- Suspected skull fracture:
  - Deformity, haemotympanum, ecchymoses around eyes or behind ears
  - Cerebrospinal fluid (CSF) leakage from ears or nose
- Post-traumatic seizure
- Focal neurological deficit
- Vomiting > once
- Amnesia for more than 30 minutes prior to injury
- Age > 65
- History of bleeding problems or any anticoagulant/antiplatelet use (recent literature suggests that anti-platelet agents may confer substantial risk of delayed bleeding)
- Severe mechanism of injury

Paediatric patients

- Witnessed loss of consciousness (LoC) for > 5 minutes
- Amnesia to events pre- or post-injury for > 5 minutes
- Abnormal drowsiness
- > 2 episodes of vomiting without alternative cause
- Post-traumatic seizure
- GCS of < 14 initially, or GCS of < 15 for infants < 1 year old (📖 GCS p. 937)
- Suspected skull fracture
  - Tense fontanelle, deformity, haemotympanum, ecchymoses around eyes or behind ears
  - CSF leakage from ears or nose
- Focal neurological deficit
- Any scalp contusion or > 5 cm laceration in child < 1 year old

• Severe mechanism of injury

Imaging the spine

Cervical spine

Up to 15% of head injured patients have a spinal injury. There are rules for cervical spinal imaging in adults (NEXUS and Canadian). Very little research exists to guide imaging the spine in children. The guidelines below serve only as general recommendations.

CT and XR are both good options, although recent literature suggests that XR will rarely miss injuries that are identified on CT. With more recent techniques, the degree of radiation exposure may be similar in the two studies. For patients who already require a CT brain, CT c-spine is the study of choice. In other patients, stability considerations may affect the choice.

Indications for imaging (XR or CT) include:

- Midline cervical spine tenderness
- GCS < 13, intoxication, and/or intubated status
- Weakness or sensory abnormalities
- Visible trauma above clavicles
- Significant distracting injury that may mask neck pain
- CT if inadequate XR views

Thoracic and lumbar spine

Both CT and XR can be used. Similar considerations as above apply. Thoracic and lumbo-sacral spinal imaging should be performed in all patients with spinal tenderness, or any neurologic deficit. Identification of any vertebral fracture mandates imaging of entire spine.

283 Blunt chest trauma

Blunt chest injuries contribute to 75% of trauma-related deaths, usually via hypoxia or major haemorrhage. Most injuries are to peripheral structures after a direct chest blow and are non-operative. Injuries to central thoracic structures are associated with rapid acceleration-deceleration forces, frequently require surgery and carry a high mortality rate. Injury to the lower thorax is often (30%) associated with liver or spleen injury.

The first five minutes

Resuscitation (see 🕮 ATLS, p. 726):

- VS, IV, O_2 by facemask, resuscitate
- Needle to mid-clavicular line, second intercostal space, for tension PTX (see Traumatic haemothorax and pneumothorax, p. 742)
- Always place chest tube (see 🕮 Procedures, p. 838) prior to positive-pressure ventilation in patients with *any* PTX to avoid conversion to tension PTX
- Cardiac tamponade: pericardiocentesis and to OT urgently – no role for front room thoracotomy in blunt trauma

History and physical examination

Key historical features

- Identify acceleration-deceleration mechanisms (MVA, fall from height) and any direct blow to the chest wall
- Pre-existing cardio-pulmonary conditions

Signs and symptoms

- Unilateral decreased breath sounds with tracheal deviation away from affected side with engorged neck veins, hypotension (suspect tension PTX)

- Hypoxia, tachypnoea
- Decreased air entry, wheeze, crepitations, pleural rub
- Flail chest, palpable rib or sternal fractures, abrasions or contusions
- Distant heart sounds, engorged neck veins, hypotension (suspect cardiac tamponade)

Possible causes and differential diagnosis

- PTX, HTX, lung contusion
- Blunt cardiac injury
- Cardiac tamponade
- Diaphragm rupture
- Flail chest or rib fractures

General investigations

- Labs: Hb ◇, troponin ◈ (more sensitive and specific than ECG for blunt cardiac injury)
- ECG ◇: (arrhythmia or inverted T-wave injury pattern in blunt cardiac injury)
- Imaging: CXR ◇ (erect PA CXR good sensitivity for PTX and effusion/HTX > 200–500 ml; supine CXR will miss up to 50% of PTX (air moves anterior), shows falsely widened mediastinum, and will show HTX as hazy opacity on affected side (veiled lung) without fluid level), US ◇ (very good sensitivity for PTX and moderate to large HTX in supine patient); CT ◈ (gold standard for PTX and HTX with lung window; with contrast, can also evaluate vascular and other tissue injuries)

Diagnosis and management of specific injuries

The goal of acute management is to restore and maintain perfusion and oxygenation and provide analgesia. Monitor for blood loss and hypoxia/hypercarbia.

Haemo-pneumothorax

See 🕮 Traumatic haemothorax and pneumothorax, p. 742 and Thoracentesis, p. 837).

Chest wall injury

NB: Children may sustain major intra-thoracic damage without fractures.

Rib fractures

- Can cause PTX
- 50% rib fractures not visible on CXR
- Visibly displaced fractures have high rate of underlying lung injury
- Aggressive pain control (nerve block, NSAIDs, opioids) and maximum inhalation exercises (use incentive spirometer when available) are essential to avoid complications of hypoventilation (atelectasis, pneumonia)

Associated high-risk injuries:

- First and second rib fractures → injury to the subclavian vessels
- Mid thoracic rib fractures → pulmonary contusion
- Lower rib fractures → liver or splenic trauma

Sternal fractures

- Direct injury to anterior chest
- High association with blunt cardiac injury
- May cause costo-chondral disruption and anterior flail chest

Flail chest (‘free’ chest wall segment)

- Multiple fractures of > 2 adjacent ribs (lateral flail)

- Fractures of > 2 ribs bilaterally (flail sternum, anterior flail)
- Rib fractures with costo-chondral disruption:
  - » Clinical diagnosis from paradoxical movement, as XR limited for cartilage
  - » Associated with severe pain, alteration of chest wall mechanics, and major underlying lung damage
- Management is based on extent of V:Q mismatch and associated injuries:
  - » Unilateral injuries without significant dysfunction managed with regional anaesthesia (intercostal, epidural or paravertebral block) and NIPPV (by bag valve mask ◇ or mechanical ⟐)
  - » Bilateral injuries or significant lung contusion managed with intubation mechanical ventilation, ⟐ and systemic analgesia

Lung parenchymal injury

Pulmonary contusion

- Initial condition deceptive, progressive over 24–48 hours with worsening hypoxia from V:Q mismatch
- CT ⟐ is the optimal diagnostic study
- PPV is usually required: NIPPV pressure support (bag valve ◇ or mechanical ⟐) for minor contusions; intubation and mechanical ventilation early ⟐ for major contusions
- Therapeutic fluid restriction is no longer recommended, though caution for volume overload until intubation and ventilation available

Tracheo-bronchial injury

- Uncommon (1–3% of major thoracic trauma) with extremely high mortality
- Most often right main bronchus within 2.5 cm of carina
- Suspect with persistent PTX after thoracostomy, severe respiratory distress, and severe subcutaneous emphysema
- CXR findings: large PTX, mediastinal gas, lung collapse onto posterior-medial diaphragm (‘fallen lung’ sign, in supine patient only, not sensitive (~20%) but pathognomonic)
- Management: selective intubation of the unaffected lung and surgical repair ⟐

Blunt cardiac injury

- Following rapid deceleration/direct blow
- Spectrum of structural damage:
  - » Minor concussion → contusion → infarction
  - » Rupture of the septum, valves and chordae
  - » Complete wall disruption
- Clinical findings depend on extent and location of damage, and range from arrhythmias to infarction to regurgitation to failure
- Management is as for any non-traumatic dysrhythmia or acute cardiac dysfunction

Diaphragmatic rupture

- Left diaphragm more than right (80% versus 20%)
- Bowel herniation acutely < 30%
- Gastric herniation may mimic PTX. *Consider before inserting chest tube!*

Critical documentation

- Serial clinical findings, including clinical response to therapy (O_2, thoracostomy, intubation, etc.)
- Ventilator settings
- Imaging and lab results, ECG interpretation
- Medications given

Disposition

Admit all symptomatic patients. For flail chest, severe lung contusion, or blunt cardiac injury – admit to ICU ◈.

284 Penetrating chest trauma

Mechanisms include stabbing, gunshot, shrapnel, and impalement injuries. The vast majority present in stable condition and will not require thoracotomy.

The first five minutes

(📖 ATLS, p. 726).

- VS, IVF, O_2 by facemask, cardiac monitor
- A: stent tracheal injury (hypoxia, blowing /sucking neck wound) with endotracheal tube (cuff distal to defect)
- B: needle to mid-clavicular line, 2nd intercostal space, for tension PTX. Follow with chest tube (see 📖 p. 838)
- Always place chest tube prior to positive-pressure ventilation in patients with *any* PTX to avoid conversion to tension PTX
- Valve dressing for sucking chest wound (see 📖 Traumatic haemothorax and pneumothorax, p. 742)
- C: *only if* cardiothoracic surgery capability available onsite, consider initiating front room thoracotomy for cardiac tamponade or witnessed loss of pulses

History and physical examination

Key historical features

- Description of weapon (including length) and mode of injury
- Unilateral or transmediastinal injury?

Signs and symptoms

- Hypoxia, blowing /sucking neck wound, or profuse bubbling from chest drain suggest major airway injury
- Over 1.5 l (or > 200 ml/h over 2 h) blood from chest drain → massive HTX
- Beck's triad (hypotension, muffled heart sounds, distended neck veins) → pericardial tamponade
- Unilateral decreased breath sounds with distended neck veins, hypotension, and contralateral tracheal deviation → tension PTX
- Chest wall or neck crepitus → PTX or airway injury
- Injury path to diaphragm or peritoneal cavity? (See 📖 Penetrating abdominal trauma, p. 748)

Possible causes and differential diagnosis

- PTX/HTX
- Cardiac/pericardial laceration (± tamponade)
- Vascular injury (aorta (see 📖 Aortic injury, p. 744), carotid, azygous, pulmonary, subclavian, vena cava, internal mammary)
- Tracheobronchial; oesophageal; diaphragm injury

Investigations

These should not delay emergency surgery when indicated:

- Labs: CBC, electrolytes, glucose, type and cross, ABG ◇
- Imaging: CXR ◇ (mark wounds to evaluate injury path, e.g. place paper clips on skin at anterior wounds and bent paper clips on posterior wounds); US (eFAST) (for PTX/HTX, haemopericardium) ◇

Specific additional investigations:

- Diaphragm injury:
  - » CT chest/ abdomen. ◈
  - » Consider diagnostic laparotomy/laparoscopy if CT not available ◇

- Airway injury:
  - » Flexible bronchoscopy ◈
- Oesophageal injury:
  - » Request water soluble (NOT barium) contrast swallow ◇
  - » Oesophagoscopy or CT chest if contrast swallow not feasible or nondiagnostic ◈
- Transmediastinal injury:
  - » CT angiogram ◈
  - » Water soluble contrast swallow ◇
  - » Oesophagoscopy ◈ if swallow study not feasible or nondiagnostic
- Cardiac injury (compensated haemopericardium):
  - » CVP >15 cm H_2O = possible pericardial tamponade ◈
- Impalement injury (with weapon in place):
  - » AP and lateral CXR to ascertain proximity to great vessels/heart ◇
  - » CT angiogram or preferably catheter-directed angiogram if available ◈

Management

The goal of acute management is to restore oxygenation and perfusion, and determine need for urgent surgery.

- Minor airway injuries (without desaturation, massive emphysema, blowing/sucking neck wound, or profuse bubbling from chest drain): wound care and observation
- Simple PTX/HTX: intercostal drain and observation
- Open PTX (sucking wound): one-way valve dressing, chest tube, then operative repair ◇ (📖 Advanced trauma life support, p. 726)
- Pericardial tamponade: emergency surgery for open repair vs. sub-xiphoid window ◇
- Oesophageal injury or diaphragm injury: urgent surgery
- Impalement: remove impaled weapon only in full-capacity operating theatre ◈

Critical documentation

Serial clinical findings, VS and other clinical response; injury mechanism and path. Document all wounds (use wound characteristics rather than 'entry' or 'exit').

Disposition

With the exception of patients with superficial chest wall injuries, admit all to ward, ICU, or OT. Patients with any abnormal VS after resuscitation should be admitted/transferred to a facility with surgical capacity.

285 Traumatic haemothorax and pneumothorax

Haemothorax (HTX) is blood in the pleural space. Massive haemothorax is defined as > 1.5 l blood in the hemithorax and is life-threatening.

Pneumothorax (PTX) is air in the pleural space: tension pneumothorax is a life-threatening manifestation in which venous return and cardiac output are compromised by increased intrathoracic pressure and mediastinal shift.

The first five minutes

Resuscitation

- ABC, VS, O_2 (📖 ATLS, p. 726).
- Evaluate and treat tension pneumothorax (needle decompression) or massive haemothorax (emergency OT thoracotomy)

History and physical examination

Key historical features

Blunt injury (with associated rib fractures) or penetrating injury (gunshot, stab wound, iatrogenic (after central line insertion)) (🕮 Penetrating chest trauma, p. 740).

Signs and symptoms

Haemothorax

- Hypoxia; tachycardia and tachypnoea, decreased chest expansion; dullness to percussion; decreased air entry on affected side
- Massive HTX > 1.5 l (or > 200 ml/h over 2 h) from chest tube

Pneumothorax

- Early findings: tachycardia and tachypnoea, chest pain, dyspnoea, anxiety; hyperresonance to percussion, subcutaneous emphysema, decreased air entry on affected side
- Late findings: hypoxia, decreased GCS, cyanosis; tracheal deviation toward the contralateral side; hypotension, distended neck veins (not present if severe hypotension)

Possible causes and differential diagnosis

- Pleural effusion
- Empyema
- Asthma: expiratory wheeze
- COPD with bullae/blebs: can be mistaken for PTX, or rupture and cause PTX
- PE: risk factors for thromboembolism, evidence of DVT
- Myocardial ischaemia: chest pressure, radiating pain, nausea, vomiting, diaphoresis
- Pleural effusion: dullness to percussion, decreased breath sounds
- Oesophageal perforation: severe retrosternal chest pain, odynophagia
- Myocardial rupture with tamponade: hypotension, distended neck veins

Investigations

A clinical diagnosis, supported by imaging; additional investigations are related to underlying cause or associated conditions.

- Imaging: CXR ◇ (HTX: supine film – white-out of the affected side, erect film – classical crescent shape on affected side; H/PTX – air-fluid level; subpulmonic HTX – elevated hemi-diaphragm, blunting of the costo-phrenic angle; PTX – supine CXR misses up to 50%, upright CXR high sensitivity); US ◇ (very high sensitivity and specificity for PTX); CT ◈ (may identify occult PTX or HTX with underlying lung contusion)

Management

The goal of acute management is to restore oxygenation. 100% O_2 (accelerates the rate of pleural air absorption), IV access, chest tube (see 🕮 Intercostal chest drain, p.), analgesia. Antibiotics are controversial.

Small pneumothorax

Observation without chest tube if: no positive pressure ventilation and no planned general anaesthetic or air transport. CXR at six hours. If no increase in size – discharge; if increase in size – chest tube.

Large pneumothorax

Chest tube. (See 🕮 Thoracentesis, p. 836.)

Open pneumothorax

- Three sided ‘open valve’ dressing (see 🕮 ATLS, p. 726). Chest tube ◇

Tension pneumothorax

• Large bore needle into second intercostal space mid-clavicular line followed by chest tube ◇

Massive haemothorax

• IVF and consider autotransfusion of output. Intubation

Critical documentation

Serial Hb and CXR; chest tube output; interventions and clinical response.

Disposition

Admit to surgery per criteria above. Admit all patients with chest tube until < 100 ml/day and no air leak (chamber bubbling) with patent tube.

286 Aortic injury

Chiefly associated with major blunt thoracic trauma, often with an acceleration-deceleration mechanism, such as motor vehicle collision or fall from height. 75–90% are fatal. Survivors often have multiple injuries. Less commonly, penetrating trauma can injure the aorta. Clinical and radiologic indicators of aortic injury in those who survive to hospital are often subtle.

The first five minutes

• ABC, VS, facemask O_2, IVF, cardiac monitor
• Aortic injury does not lead to hypotension unless actively bleeding – rule out other sources in the hypotensive patient

History and physical examination

Key historical features

• Mechanism suggestive of aortic injury

Signs and symptoms

Usually very limited clinical findings:
• Other blunt thoracic injury – lung contusion, multiple rib fractures, flail chest
• Differential BP, higher in upper than lower limbs
• Pulses show radio-femoral delay
• May present with hypertension or hypotension, the latter usually from other injuries

Investigations

Facility with advanced imaging capability recommended; clinical findings are unreliable.
• Labs: Hgb, type and cross ◇
• Imaging: CXR ◇ (widened mediastinum (> 8 cm at T4); NGT displaced to the right; right main bronchus displaced upward; left mainstem bronchus displaced downward; loss of the aortic knuckle; left sided pleural capping (extrapleural haematoma, left sided haemothorax); contrast enhanced CT-angiogram ◈ (examination of choice); catheter-directed angiogram ◈ (technical gold standard)

Possible causes and differential diagnosis

• Other vascular injury with haematoma
• Non-traumatic causes of wide mediastinum on CXR (thymus gland, intrathoracic thyroid, lymphoma etc.) – CT

scan can distinguish

Management

The goal of acute management is to stabilise and resuscitate symptomatic patients and identify aortic injury to facilitate early referral and repair.

Initial medical management

- Control pain and BP at MAP of 65–70 mmHg, with beta-blocker if tolerated
- Manage other injuries
- Transfer to facility with vascular/thoracic surgery

Definitive management

- Majority of cases will undergo endovascular stent graft repair once stabilised
- Some may require complex open thoracic or vascular repair on bypass

Critical documentation

Careful description of mechanism and associated injuries; serial VS, including upper and lower extremity pulses; serial exams; radiography interpretation.

Disposition

Transfer to a major trauma facility with thoracic and/or vascular surgery, and, where possible, endovascular interventional capability, with continuous monitoring and via advanced life support transport if available.

287 Blunt abdominal trauma

Intraperitoneal injury is one of the most difficult conditions to exclude in polytrauma patients. Physical exam is notoriously limited and may miss up to 50% of injuries. Ultrasound (FAST) may be helpful to identify intraperitoneal fluid, especially in hypotensive patients, but cannot rule out intraperitoneal fluid or injury. All patients with abdominal pain or visible evidence of abdominal trauma should undergo serial exams over several hours, even with normal imaging.

The first five minutes

- ABC, IV access
- Consider abdomen as source of bleeding in all hypotensive patients
- Consider bowel injury in all polytrauma patients presenting with systemic inflammation

History and physical examination

Key historical features

- Direct blunt force produces crush injury of solid or hollow organ against spine, pelvis or abdominal wall
- Deceleration forces (MVA, ejection, fall from height) produces sudden increase in intra-luminal bowel pressure leading to rupture
- Vomiting (blood?)

Signs and symptoms

- Inspection: abrasions, contusions, flank (Grey-Turner's sign) and umbilical discolouration (Cullen's sign)
- Abdominal tenderness (note that lower rib, pelvis, and chest injuries, or head injury with AMS, may interfere with assessment of abdominal tenderness)
- Rigid, severe or increasing tenderness to palpation = peritonitis

- Lower rib or pelvic fractures can cause organ or vessel laceration
- Frequent association with chest injury (truncal injury pattern)
- Referred pain: left shoulder (Kehr's sign) with splenic injury; right shoulder with liver injury (head lowered and legs elevated to elicit these)
- Auscultation: silent abdomen suggests ileus (associated with peritonitis)
- Seat-belt/tyre marks: 20% have intra-abdominal injuries (consider also fracture of lumbar vertebrae)

Possible causes and differential diagnosis

- Injury of hollow or solid viscera, vessels or genitourinary tract

Investigations

- Labs: Hgb ◇ (monitor for ongoing bleeding with non-operative solid organ injury); amylase ◈ (of limited utility)
- Imaging: FAST/eFAST; CT ◈ in stable/resuscitated patients (may miss hollow organ injury, especially if done early); CXR (exclude free air) ◇; AXR (and pelvis XR) (pelvic or spinal fractures/retroperitoneal gas, loss of psoas shadow)
- Diagnostic peritoneal aspiration of all four quadrants ◇ for haemodynamically unstable patients: takes one minute, > 10 cc blood indicates need for transfer to facility with surgical capacity – when indicated, do not delay laparotomy for other investigations
- DPL ◇: open or closed technique, with microscopy, if bleeding suspected and unknown source
- Evaluate renal tract (see 🕮 Genitourinary trauma, p. 752)

Management

The goal of acute management is to stabilise haemodynamics and determine whether urgent laparotomy is required.

Hollow viscus (bowel, stomach) injuries are necessarily surgical conditions, and morbidity increases rapidly if surgery is delayed. Some solid organ (spleen, liver) injuries in stable patients may be appropriate for observational management.

- IV morphine ◇
- Serial exams Q15–30 minutes
- Monitor serial Hgb Q4-6h ◇
- Indications for emergency laparotomy in patients with abdominal trauma include:
  - » XR or CT evidence of pneumoperitoneum (hollow viscus injury)
  - » Persistent evidence of peritoneal irritation
  - » DPA/DPL positive in hypotensive patient
  - » Persistent, significant GI bleeding in NGT or vomitus
  - » Persistent unexplained hypotension

Critical documentation

History, serial clinical findings, response to resuscitation, imaging and laboratory results.

Disposition

Admit all patients with significant abdominal trauma – observe for occult injury. Refer all symptomatic patients urgently to a facility with surgical capacity.

288 Penetrating abdominal trauma

Penetrating abdominal trauma will often require emergency laparotomy. Hollow viscus (bowel, stomach) injuries are necessarily surgical conditions, and morbidity increases rapidly with delay to surgery. Some solid organ (spleen, liver) injuries in stable patients may be appropriate for observational management.

The first five minutes

- ABC, VS, IVF (see 🕮 ATLS, p. 726)
- Consider bowel injury in all patients presenting with abdominal pain, peritonitis or systemic inflammation
- Consider injury trajectory and thoracic cavity involvement (see 🕮 Penetrating chest trauma, p. 740)

History and physical examination

Key historical features

- Stab wounds and impalements: length of weapon/object
- Gunshot wound (GSW): weapon, number of shots, low or high velocity?

Signs and symptoms

- Signs of shock
- Signs of peritonitis (percussion is best test for peritonitis, *not* rebound):
  - Tenderness distant from wound
  - Abdominal distension
  - Systemic inflammatory response (fever, tachycardia, hypotension, etc.)
- Rectal and vaginal exam for bleeding, or wounds
- Eviscerated bowel

Possible causes and differential diagnosis

- Injury to bowel, solid organs, pancreas, urinary tract or vascular system

Investigations

- Labs: electrolytes, CBC, type and cross ◇, PT/PTT ◈
- Imaging: CXR ◇ (mandatory, erect or lateral abdominal shoot-through); AXR ◇ (mark wounds prior to XR injury path – e.g. place paper clip on skin at anterior wounds and bent paper clip at posterior wounds; do AP and lateral, not supine and erect!); CT scan ◈ (may be done for stable patients, though DO NOT delay indicated laparotomy); US (FAST) ◈ (may identify free peritoneal fluid, but unlikely to change management, which is driven by clinical findings)

Management

The goal of acute management is to stabilise haemodynamics and determine whether urgent laparotomy is required.

- Resuscitation (see 🕮 ATLS, p. 726)
- Absolute indications for laparotomy (if no OT, give broad-spectrum antibiotics pending transfer to surgical facility):
  - Haemodynamic instability
  - Peritonitis or drainage of faecal content
  - CXR with free air
  - Abdominal GSW
  - Evisceration of bowel (if transfer required: replace bowel within abdomen; apply moist sterile dressing and occlusive cover and stabilise so there is no gravitational pull on bowel segments)
- In haemodynamically stable patients, the following may be observed on a surgical service with immediate OT availability:
  - Abdominal stab wounds with none of indications above
  - Isolated omental evisceration with none of indications above

Critical documentation

- Initial clinical findings, including identification and description of all wounds (use wound characteristics rather than ‘entry’ or ‘exit’)
- Response to resuscitation

- Serial VS and abdominal exams
- All imaging and laboratory results

Disposition

With the exception of patients with superficial abdominal wall injuries, admit to ward, ICU, or OT.

289 Pelvic trauma

Pelvic fractures are an important cause of haemorrhagic shock in the major trauma patient, due to associated disruption of pelvic vasculature. Stabilisation requires a rapid multimodal approach, including simple mechanical measures to achieve haemostasis and early referral for operative and/or angiographic interventions. Often associated with serious injuries elsewhere.

Classification

Pelvic fractures can be classified by the pattern of force and identified radiologically by the site of factures. There is an association between the different types of fractures and the risk of haemorrhagic shock.

Lateral compression

- Internal rotation of hemipelvis
- Compression of pelvic volume
- Life-threatening haemorrhage uncommon

Antero-posterior compression (‘open book’)

- Disrupts symphysis pubis, often with sacroiliac fracture/dislocation
- Opening of pelvic ring
- Potential for major haemorrhage

Vertical shear

- Disruption of sacrospinous and sacrotuberous ligaments
- Major pelvic instability
- High risk of life-threatening haemorrhage

In addition, avulsion fractures may occur at the anterior inferior and superior iliac spines, and at the ischial tuberosity, but are not associated with pelvic instability or vascular disruption.

The first five minutes

- ABC, VS, O_2 by face mask, IVF (see 🕮 ATLS, p. 726)
- Unexplained hypotension; assume pelvic instability until XR excludes significant fractures. Manipulation may cause clot disruption and further injury
- Splint unstable pelvis with purpose-made fixation device or bedsheet tied around hips, spanning from iliac spine to inguinal crease. *Do not* log roll patients with unstable pelvic fractures

History and physical examination

Key historical features

Mechanism of injury: high-risk include MVA (pedestrian or occupant), fall from height, crush injury.

Signs and symptoms

Additional clinical features of a pelvic fracture may include evidence of a ruptured urethra (scrotal haematoma,

blood at urethral meatus), limb length discrepancy or a rotational deformity of the lower limb.

- Pelvic instability
- Tenderness on palpation or pain on lower limb movement
- Injuries to urethra, bladder, rectum and genitalia: blood at external meatus, scrotal haematoma, frank haematuria, PR or PV blood
- Evaluate all patients for intraperitoneal injury, as source of pain and tenderness may be difficult to distinguish on exam

Investigations

- Lab: Hgb, type and cross ◇, PT/PTT ◈
- Imaging: pelvis XR ◇ (mandatory in all major trauma patients); pelvis CT ◈ (may confirm injuries not identifiable on XR, including active extravasation from the pelvic vasculature; should not delay resuscitation and indicated operative interventions); retrograde urethrogram ◈ (must be performed if there is suspicion of urethral injury (blood at external meatus, scrotal haematoma)); in the absence of urethral injury, a urinary catheter insertion can be attempted once; if resistance, bleeding or failure to pass on first attempt, then insert suprapubic catheter (see 🕮 Catheters, p. 822 and p. 824)

Management

The goal of acute management is to stop bleeding and resuscitate.

- O_2, two large-bore IV lines, rapid IVF
- Application of a sheet or pelvic binder as above (internally rotates the lower limbs, reducing the pelvic volume)
- Emergency external fixation ◈, laparotomy for packing the pelvis ◇ and/or angiographic embolisation ◈ may be required
- Avulsion fractures may be managed with analgesia and bed rest

Disposition

Admit haemodynamically stable patients to surgery service ◇. Unstable patients should be transferred/admitted directly to the nearest operating theatre.

Patients with simple isolated avulsion fractures and no concern for other injury may be discharged with analgesia, bed rest, and close follow-up.

290 Genitourinary trauma

Genitourinary tract (GUT) trauma occurs in about 10% of all injured patients. All females with GUT trauma require a vaginal speculum examination. Straddle injuries are a common cause of genital and urethral injuries in children.

The first five minutes

- ABC, VS, control bleeding, stabilise patient, analgesia (see 🕮 ATLS, p. 726)
- Degloved penis or scrotum: cover with warm saline dressing

History and physical examination

Key historical features

- Mechanism of injury (blunt, penetrating, straddle)
- Urination since injury, haematuria

Signs and symptoms

Flank haematoma or mass coupled with haematuria (macroscopic or microscopic) or a distended bladder should raise the likelihood of GUT injury. Consider associated urethral injury/transection in patients unable to urinate after genital trauma, especially straddle injury.

- Renal injury:
  - Flank abrasions/ecchymosis, pain/tenderness
  - Fractured lower ribs
  - Penetrating injury
  - Abdominal tenderness
- Ureter injury: history of penetrating trauma; may be asymptomatic
- Bladder injury: macroscopic haematuria; suspect in pelvic fracture
- Urethral injury:
  - Fresh blood at meatus (most anterior tears)
  - Haematuria with first voiding that clears
  - Inability to urinate
  - Haematoma/swelling of perineum and scrotum
  - High-riding prostate is an unreliable sign
- Penile injury:
  - Injuries usually visible on inspection and include laceration/degloving, amputation, fracture (rupture of corpus cavernosum)
  - Deviation of penis ± associated haematoma → penile fracture
- Testicular or scrotal injury:
  - Visible oedema, bruising, laceration
  - Dislocation of testicle from scrotal sac
  - Crush injury with evidence of superficial trauma → consider underlying testicular fracture
- Vaginal/vulvar injury:
  - Vaginal bleeding: lacerations may be visible only on speculum exam
  - Vaginal wall mass → haematoma
  - Vulvar mass (haematoma may be several cm in diameter)

Investigations

Single shot intravenous pyelogram (IVP) no longer advocated.

- Penetrating trauma – CT IVP ◈
- Blunt trauma:
  - Microscopic haematuria: no further investigations
  - Macroscopic haematuria: retrograde urethrogram (RUG), cystogram ◇, CT-IVP ◈
- Bladder US in patients unable to urinate ◇
- eFAST for free fluid or to compare with contralateral kidney ◈
- Testicular injuries should be evaluated with ultrasound for haematoma, fracture, dislocation

Management

The goal of acute management is early identification of GUT injury, haemorrhage and pain control, and early operative management as indicated.

Unstable patients

Resuscitate (see 🕮 ATLS, p. 726). Refer to surgery for operative treatment.

Renal injury

- Bleeding pedicle injuries require nephrectomy
- Non-operative management of non-bleeding renal injuries is an option
- Non-perfusion of the kidney on imaging = dead kidney
- Penetrating injuries tend to be more severe
- CT injury grading determines management

Indications for surgery:

• Unstable patient
• Expanding pulsatile hematoma
• Grade 5 renal injury (no blood flow to kidney)
• Extravasation of contrast is controversial and can be treated non-operatively

Non-operative management: low failure rate, but higher complication rate. Admission and regular follow-up.
• Early complications: bleeding, infection, hypertension, urinoma
• Delayed complications: bleeding, hydronephrosis, hypertension, pseudoaneurysm

Ureteric injuries

Uncommon – 1% of all GUT injuries. Require surgical repair:
• Often delayed with gunshot wounds due to later perforation
• May not have haematuria
• Rare in blunt trauma

Bladder injuries

Strong association with pelvic fractures. Bladder injuries can be classified as:
• Extraperitoneal: may heal with catheter, antibiotics
• Intraperitoneal: require open surgical repair

Urethral injuries

Mostly blunt trauma in males and rare in females: 90% due to pelvic fractures.
• In unstable patients: *single* gentle catheter attempt is unlikely to cause more damage. If attempt fails: suprapubic catheter
• In stable patients RUG prior to catheter insertion

Genital injuries

• Evaluate all patients, especially children, for non-accidental trauma and sexual assault
• Early urological consult for testicular injury as crush, fracture, dislocation, and haematoma require operative intervention
• Allow limited vulvar lacerations to heal by secondary intention (do not suture)
• Vaginal lacerations may need to be repaired under anaesthesia and expanding vaginal wall haematomas may require incision and small vessel ligation

Critical documentation

Serial exams, results of imaging studies, interventions.

Disposition

Admit all unstable patients to OT; refer all patients to urology or surgery.

291 Head injuries

Head injury is extremely common. The skull is a rigid container and clinical condition may deteriorate unpredictably with increased intracranial pressure (ICP) from brain bleeding or oedema, so close monitoring and serial exams are essential.

The first five minutes

• ABC, including intubation if unable to protect airway (see 🕮 ATLS, p. 726)
• Assume cervical spine fracture and immobilise
• Check glucose in all patients

History and physical examination

Key historical features

Mechanism, loss of consciousness (duration – intermittent?), drug or alcohol use, anticoagulant use (aspirin, warfarin, clopidogrel, heparin).

Signs and symptoms

- Visual changes, amnesia, seizures, vomiting, headache
- GCS, VS (Cushing's response – bradycardia and hypertension – suggests increased ICP and may signal impending cerebral herniation)
- Pupils: sluggish reactivity or unequal size raises concern for herniation; bilaterally fixed dilated pupils indicate very poor prognosis
- Scalp exam for lacerations, depressed skull fractures and haematomas
- Signs of basilar skull fracture: raccoon/panda eyes (ecchymosis around orbits), Battle's sign (ecchymosis behind ears), haemotympanum, rhinorrhoea, otorrhoea

Possible causes and differential diagnosis

- Diffuse axonal injury (DAI) – often devastating, cause of persistent vegetative states, typically not seen on CT (MRI more sensitive)
- Concussion – mild DAI, no bleeding, normal CT
- Intracranial haemorrhage
  - » Subdural haematoma (SDH, tear bridging veins)
  - » Epidural haematoma (EDH, laceration of meningeal artery)
  - » Subarrachnoid haemorrhage (SAH, shearing of subarachnoid arterioles)
  - » Intraparenchymal haemorrhage (IPH, cerebral contusion)

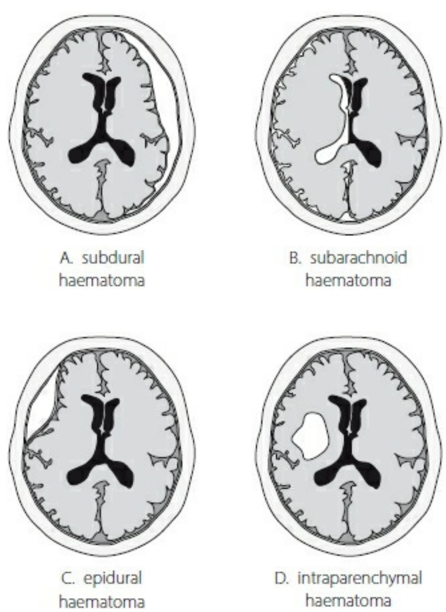

Figure 291.1 A subdural haematoma; B subarrachnoid haemorrhage; C epidural haematoma; D Intraparenchymal haemorrhage

Investigations

- Labs: Hgb, glucose, type and crossmatch

- Imaging: patients with GCS <13 should undergo transfer for CT ◈ (see 🕮 Approach to neuro-imaging, p. 734)

Management

The goal of acute management is to prevent hypoxia, hypotension, and excessive ICP to avoid secondary brain injury. Avoid hypotension (SBP < 90 mmHg) and hypoxia (spO_2 < 92%): can increase mortality by 150%.

- Continuous/frequent monitoring of VS
- If no access to CT, and signs of herniation, consider burr hole craniotomy (See 🕮 Burr holes, p. 854)

Scalp lacerations

Irrigate and close early to prevent excessive blood loss. Check tetanus coverage (🕮 p. 362).

Skull fracture

Management is controversial – guided by neurosurgery. Antibiotic prophylaxis for vault fractures, but not skull base.

ICP reduction

- Elevate head of bed to 30 degrees
- Mannitol IV 0.5–1 g/kg (temporising, use only if neurosurgery pending)
- DO NOT give steroids
- Sedation and analgesia can lower ICP. Mechanically ventilate if severe TBI ◈; aim for PCO_2 of 35 mmHg (3.5–4.5 kPa)

Critical documentation

Serial neurologic exams, including GCS, and motor, sensory, and cranial nerve findings.

Disposition

- Transfer for CT all patients with GCS <13; refer to neurosurgery based on results
- Mild TBI (GCS 13–15) – evaluate at any level of facility; patients with LoC or abnormal neurological examination should be observed for 48 hours if CT is not done
- Discharge patients with a GCS of 15, without prolonged LoC, and a normal neurological examination (with someone at home to monitor for 48 hours)

292 Approach to penetrating neck trauma

Defined by breach of platysma muscle, penetrating neck injuries may compromise vital structures (including the larynx and trachea, oesophagus, lung, major vessels, cranial nerves and spine) by direct injury or compression from nearby haematoma. Life-threatening injuries may be occult, and missed injuries result in major morbidity and mortality.

The first five minutes

- VS, IV, O_2 by facemask (see 🕮 ATLS, p. 726)
- A: secure airway early. Control airway bleeding
- Bubbling wound, haemoptysis, voice change, suggest laryngotracheal trauma: firm manual compression to reduce air leak may improve oxygenation. Orotracheal intubation ◇ with cuff distal to defect
- Cricothyroidotomy ◇ if unable to intubate (🕮 p. 862)
- Major open larnygotracheal wounds, intubate the distal transected segment under direct visualisation, grasping with tissue-holding forceps ◇
- B: needle to mid-clavicular line, 2nd intercostal space, for tension PTX – follow with chest tube (see 🕮 Intercostal chest drain, p. 838) – place chest tube prior to positive-pressure ventilation in patients with any PTX

to avoid conversion to tension PTX
• C: consider occult bleeding into pleural cavity (massive HTX); do not probe wounds beyond need for haemorrhage control
• D: spinal cord injury, brain injury due to poor perfusion or embolic complications of vascular injury

History and physical examination

Key historical features

Description of weapon (including length) and mode of injury.

Signs and symptoms

• Expanding haematoma
• Hypoxia, blowing/sucking wound (tracheal injury)
• Crepitus (airway injury, PTX)
• Carotid bruit or palpable thrill (vascular injury)
• Median, radial, ulnar nerve deficit (brachial plexus injury)
• Other neurologic deficits (localise lesion to brain, CN, Horner's syndrome, or spinal cord).

Anatomy

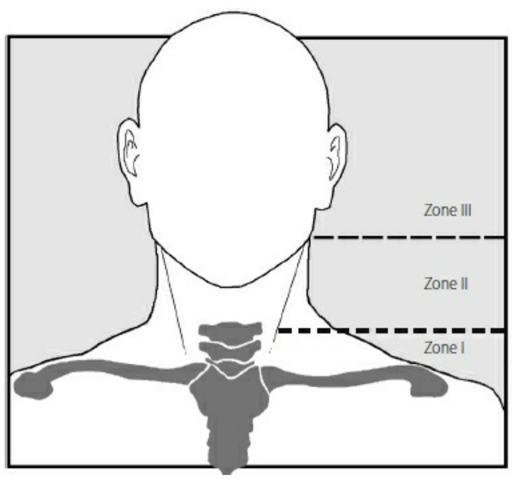

Figure 292.1 Zones of the anterior triangle of the neck

• Zone I: clavicle to cricoid:
  » Innominate, common carotid, subclavian vessels and vertebral artery, brachial plexus, trachea, oesophagus, apex of the lung, and thoracic duct
• Zone II: cricoid cartilage to angle of the mandible:
  » Carotid and vertebral arteries, IJV, larynx and pharynx
• Zone III: above the angle of the mandible to skull base
  » Distal carotid, vertebral arteries and pharynx
  » Poorly accessible to physical examination

Possible causes and differential diagnosis

• Injury of airway, vascular, oesophageal, neurological

Investigations

• Labs: Hb, type and cross ◇

• Imaging: lateral neck XR ◇ (prevertebral air (aerodigestive injury), prevertebral tissue swelling, tracheal deviation, bony injury); CXR ◇ (thoracic inlet or mediastinal contour abnormality (vascular injury); pneumomediastinum (aerodigestive injury or PTX); pneumopericardium (cardiac injury); PTX/HTX; raised hemidiaphragm (subpulmonic HTX, phrenic nerve injury)); CT angiogram ◈ (investigation of choice in stable patients; combine with contrast swallow or endoscopy to rule out oesophageal injury)

Table 292.1 Clinical features that dictate specific investigations ◈

	Clinical manifestation	Investigation
Vessels	Expanding or pulsatile haematoma Bruit / thrill Peripheral pulses	CT angiography
Larynx – trachea – oesophagus	Haemoptysis Air bubbling through wound Subcutaneous emphysema Dysphagia /odynophagia Hoarseness	Contrast swallow / endoscopy Laryngoscopy Pharyngoscopy
Nervous system	GCS Localising signs Spinal cord Cranial nerves Brachial plexus (median, radial, ulnar) Horner's syndrome	CT brain CT spine MRI

Management

• Consider early fibre optic intubation for large midline haematoma even in stable patient ◈
• No IV on side of neck wound
• Trendelenburg position to reduce risk of air-embolism with venous injury
• Consider Foley-catheter tamponade. Insert catheter into wound until resistance is felt, inflate balloon with saline/water until bleeding arrested or resistance encountered, may use more than one catheter. Clamp central lumen

Critical documentation

• Serial clinical findings, including VS, vascular and neurological status; clinical response to interventions
• Injury mechanism and path. Document all wounds (use wound characteristics rather than 'entry' or 'exit')
• Imaging results

Disposition

Admit all patients to surgery.

293 Maxillofacial trauma

Maxillofacial injuries are common and can cause early mortality from airway compromise or blood loss (the face is highly vascularised and blood loss can be substantial) (📖 Eye trauma, p. 764).

The first five minutes

• ABC; secure airway and control epistaxis and other bleeding early (temporary suture if needed), resuscitate, IVF as needed
• Evaluate need for C-spine immobilisation
• Allow patient to sit forward or tilt backboard to keep blood out of the airway. Do not remove impaled objects
• Remember head trauma evaluation; check glucose if ALoC

History and physical examination

Key historical features

- Mechanism: direction of blow to face, fall, object causing penetrating wound
- LoC (duration), drug or alcohol use, anticoagulants (aspirin, warfarin, clopidogrel)

Signs and symptoms

- Intra-cranial injury: amnesia, seizures, vomiting, headache, visual changes
- Any bite mismatch? Able to breathe through both sides of nose?
- Check motor (CN VII) and sensory (CN V1, V2, V3)
- Nasal: bridge tenderness, deformity, periorbital bruising, epistaxis. Check for CSF rhinorrhoea
- Mandible: trismus, malocclusion, lower lip numbness, visible deformity, visible mandibular separation, tongue blade test (unable to bite and hold tongue depressor tightly between molars as blade is twisted to breaking) – usually fracture, and fractures usually multiple
- Mid-facial fractures: if unable, mobile midface/hard palate, crepitus (sinus or open fractures), step-off, tenderness, anaesthesia, motor and sensory defects (injury to facial or trigeminal nerve), often defy classic patterns, airway compromise, malocclusion
- TMJ: malocclusion, jaw protrudes, tender over TMJ or temporal fossa
- Malar complex: tender over zygoma, maxilla and ipsilateral orbit
- Orbital wall fractures: (see 🕮 Eye trauma, p. 764).

Possible causes and differential diagnosis

- Nasal fracture
- Mandibular fracture
- Malar complex fracture
- Mid-facial fractures (Le Fort I – III) (see Figure 293.1)
- Orbital blow-out
- Le Fort I: low-level fractures above the roots of the upper teeth
- Le Fort II: a pyramidal fracture, which may be unilateral. Disjunction is detected at infraorbital region and there may also be a midpalatal fracture
- Le Fort III: craniomaxillary disjunction, with superior fractures running above the nose, involving the frontal sinus occasionally and leading to elongation of the face. The orbital extent of the fracture may vary enormously. Palatal fractures are seen

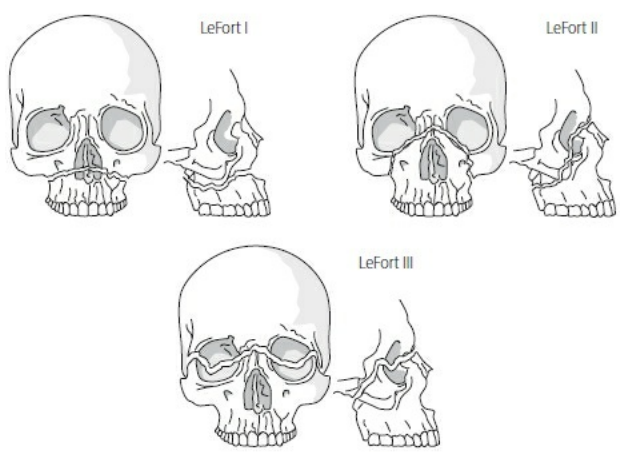

Figure 293.1 Le Fort fractures of the midfacial skeleton

Investigations

Imaging:

- Mandible/TMJ: orthopantogram, condylar view radiographs ◇
- Facial: waters view radiograph – sinus opacity ◇
- Facial: facial CT with bone windows and 3D reconstruction ◈
- Ongoing bleeding: angiogram ◈

Management

The goal of acute management is to secure the airway and stop haemorrhage:

- Intubate early and stop bleeding with nasal packing if needed
- Treat open fractures, oral cavity wounds or CSF leak with antibiotics (usually beta-lactams, or clindamycin to cover anaerobes)
- Nasal:
  - » Immediate reduction for displaced simple nasal fractures presenting within several hours
  - » Immediately drain and pack septal haematoma (septal necrosis leading to bridge deformity if untreated). Haematoma incision converts fractures to open – give antibiotics
- Mandible:
  - » Control bleeding, clean wounds, soft diet, refer as needed
  - » Antibiotics, pain control
  - » Emergency maxillofacial or dental consult ◇
- Midface/malar complex:
  - » ABCs, clean wounds, refer
  - » Antibiotics, analgesia
  - » Operative haemorrhage control if needed, surgical repair ◇ and consider angio-embolisation ◈

Critical documentation

Document visual acuity, cranial nerve examination and neurovascular status before and after any intervention.

Disposition

Admit patients with airway compromise, significant blood loss, neurovascular compromise or injuries that require surgery, preferably to plastic or maxillofacial surgery.

294 Approach to eye trauma

A systematic ocular exam (Emergency eye examination, p. 508) is essential in all trauma patients, especially those with facial fractures. Ocular injuries are often overlooked in polytrauma patients, especially those with AMS. Evaluate all eye trauma patients for intracranial injury. The occulocardiac reflex can cause bradycardia and vomiting, especially in children.

Key historical features

- Mechanism – blunt, penetrating, or chemical
- Baseline visual acuity
- If any possibility of high-velocity fragments (explosion, sanding or grinding work, flying glass), evaluate for globe penetration

Specific injuries

Orbital wall fractures

Complications include intraocular injury, sinus fracture, injury to lacrimal duct system, extra-ocular muscle entrapment:

- Nasoethmoid (medial rectus entrapment, injury to medial canthal ligament and lacrimal duct)
- Orbital zygomatic: (high impact lateral blow, often associated floor fracture)

- Orbital floor: inferior rectus entrapment
- Orbital roof: more common in children; high rate of intracranial injury

Retrobulbar haematoma (postseptal haemorrhage)

Examine for proptosis. Measure intraocular pressure (IOP) ◈. Lateral canthotomy is indicated for acute visual loss, increased IOP, and proptosis, OR in unconscious patient, if IOP > 40 (or globe feels harder than normal eye – through closed lid – if no tonometry) (📖 Lateral canthotomy, p. 834).

Vitreous haemorrhage

Results from blunt trauma causing bleeding in retina and uvea. Look for floaters (dark spots moving in visual field). There is a diminished red reflex. Use ocular US in B-scan mode to evaluate for retinal injury. Elevate the head of the bed. Avoid valsalva manoeuvers and blood thinners. Seek an ophthalmologic consultation.

Lens subluxation and dislocation

Results from blunt trauma causing disruption of lens. Causes monocular diplopia (double vision in one eye only) or visual changes. Edge of dislocated lens visible when pupil dilated. If subluxation, observation may be appropriate. If dislocation, ophthalmologic surgical management.

Extraocular muscle entrapment (prolapse into orbital wall fracture)

Presents with restricted upward or lateral gaze, orbit/cheek deformity or emphysema, endophthalmos. Needs surgical correction.

Globe penetration

Caused by high-velocity fragment or sanding/grinding history. Rust ring often present. Seidel test (slit lamp examination with cobalt blue light, positive fluorescein stream) for occult rupture. XR eye ◇ or CT orbits for foreign body (FB) detection ◈.

Globe rupture

Causes a flat anterior chamber, with teardrop pupil. Needs emergency surgery. Place eye shield to protect eye from any pressure. Do not measure IOP.

Hyphaema

📖 Hyphaema, p. 766.

Corneal abrasion and foreign body

📖 Approach to the red eye, p. 511.

Eyelid laceration

Consider underlying globe injury. Repair small superficial eyelid lacerations. Specialist repair indicated if: involves lid margin, within 6–8 mm of medial canthus, involves lacrimal duct or sac, involves inner surface of the lid, ptosis present, or involves tarsal plate or levator palpebrae muscle.

Chemical exposure

Alkali is worse than acid. Check pH repeatedly during copious irrigation. Continue until pH neutral (7.0). If pH paper not available, irrigate with at least 1–2 L fluid over 30 minutes. Emergency ophthalmologic evaluation.

Retinal detachment

Presents with sudden painless vision loss (floaters, shadows, or ‘curtain falling’) due to separation of neurosensory retina from epithelial/choroid layer. Diagnose by ocular US. Emergency ophthalmology consult for possible repair.

Subconjunctival haematoma

📖 Approach to the red eye, p. 511.

Critical documentation

Document mechanism of injury and all vital signs of the eye.

Disposition

Admit as per underlying injuries or undertake emergency ophthalmology consultation as detailed above.

295 Hyphaema

Hyphaema is the presence of blood in the anterior chamber of the eye. It usually results from trauma. Complications may be increased when associated with medical conditions such as sickle cell disease and coagulopathies. Hyphaema can lead to permanent vision loss if haeme pigment staining of the cornea crosses the visual axis. Microhyphema refers to dispersed red cells in the anterior chamber that do not layer out to form a grossly visible fluid level.

The first five minutes

Resuscitation as needed (see 📖 ATLS, p. 726).

- Place patient in a dim room; bed rest; instruct them not to read anything; elevate head of bed 45 degrees; place an eye shield. Goal is to limit eye movement to prevent further bleeding
- Evaluate for head injury, other trauma, or other bleeding

History and physical examination

- Mechanism of injury; pain, photophobia, blurred vision, headache, nausea and vomiting; medical history (sickle cell disease, bleeding disorders and anti-clotting medication, e.g. warfarin)
- Slit lamp shows blood in the anterior chamber and may show corneal blood staining with golden discoloration; measure blood layer in the upright patient as percentage of anterior chamber area
- Measure intraocular pressure – contraindicated in suspected globe rupture
- Grade severity (while upright) (Table 295.1)

Table 295.1 Grading the severity of a hyphaema

Grade	Area of anterior chamber covered with blood	Best prognosis for 6/18 vision or better
Microhyphaema	Circulating red blood cells by slit lamp examination	90%
I	< 33%	90%
II	33% –50%	70%
III	> 50%	50%
IV	100%	50%

Investigations

Investigations may elucidate underlying causes.

- Labs: CBC ◇, PT/PTT ◈ as indicated
- Imaging: CT orbit without contrast for associated injury ◈

Management

The goal of acute management is to reduce IOP, prevent rebleeding, minimise corneal staining, and treat pain and

nausea.

General:

- Instil one drop of cyclopentolate 1% to dilate pupil for examination and for pain relief. Avoid in globe rupture
- Analgesia – topical local anaesthetic drops. Avoid anti-platelet agents (NSAIDS, aspirin)
- Give antiemetics as needed
- Consult ophthalmology
- Consider eye shield and limit reading

Prevention of secondary bleeding:

- Topical glucocorticoids – one drop of prednisolone acetate 1% or dexamethasone sodium phosphate 0.1% QID ◇
- Antifibrinolytic lysine analogues (aminocaproic and tranexamic acid) are controversial – consider in healthy individuals with traumatic hyphaema ◈

Maintain normal intraocular pressure:

- Use agents that suppress aqueous flow, such as carbonic anhydrase inhibitors (acetazolamide, 500 mg TID) and beta-blockers (timolol 0.5% drops)

For patients who do not improve despite maximal medical therapy, surgical clot removal may be necessary.

Critical documentation

Mechanism or risk factor, initial visual acuity, grade of hyphaema, medication and supportive care, serial visual acuity.

Disposition

Admit if: associated with injuries that need hospital care, bleeding disorders, sickle cell disease, intraocular hypertension; suspected non-accidental injury (NAI); delayed presentation, large hyphaema (Grade III), or receiving systemic antifibrinolytic therapy .

296 Spinal injury

Spinal cord injury (SCI) affects 40 million people each year, and often results from MVC. Complete SCI is characterised by complete motor and sensory loss below the level of the lesion. Partial injuries are characterised by mixed motor and sensory findings, and partial sparing may be subtle (preserved peri-anal sensation). Deficits from complete cord injuries are irreversible, while partial injuries may be associated with substantial recovery.

The first five minutes

- ABC, VS, O_2, in-line stabilisation or triple immobilisation (C-collar, head blocks, spine board) of the cervical spine (see 🕮 ATLS, p. 726)
- Basic CNS exam prior to paralytic agents
- Immobilise unconscious patients until injury can be excluded
- Consider neurogenic shock in patients with hypotension not responsive to volume resuscitation, and a relative bradycardia

History and physical examination

Key historical features

- Mechanism and time of injury: onset and progression of symptoms
- Any alcohol or drug use
- High-risk features on history:
  - Age > 65
  - Non-ambulatory since injury

» Dangerous mechanism: fall from > 3 m, axial load, MVC with speed > 100 km/hr or rollover, motorbike, bicycle collision
» AMS

Signs and symptoms

• Midline spinal pain/tenderness, neurologic abnormalities. Motor: paralysis, decreased power or tone, absent anal sphincter reflex, loss of bladder and bowel control. Sensory: paraesthesia, dysaesthesia or numbness. Autonomic: flushing, labile BP, priapism (associated with high cord injuries)
• Clinical spinal cord syndromes:
» Brown-Séquard (penetrating injury or severe hyperflexion/extension with lateral hemi-transection of cord): loss of power and light touch ipsilateral, and loss of pain and temperature sensation contralateral
» Anterior cord syndrome (often hyperflexion): loss of power and pain sensation distal to lesion with preserved position, light touch, and vibration sense (dorsal column sparing)
» Central cord syndrome (often hyperextension, cervical region): motor loss arms > legs, variable sensation loss, ± urinary retention
» See also 🕮 Cauda equina syndrome, p. 458.

Possible causes and differential diagnosis

• Intracranial injury
• Spinal column bone, muscle or ligamentous injury
• SCIWORA (spinal cord injury without radiographic abnormality)

Investigations

• Imaging: lateral C-spine XR (C1-T1) ◇; spinal XR (AP, lateral and open mouth); Swimmer's view (if lateral inadequate – top of T1 not visible); CT or MRI ◈ if XR inadequate or non-diagnostic
• With high-risk features (see above), spine can only be cleared on negative imaging AND absence/resolution of symptoms
• Note that ligamentous injury without fracture may create an unstable column, putting the cord at risk. This may be detectable on XR as mis-alignment without obvious fracture; if XR negative, all patients with neurologic abnormalities should be investigated further

Management

The goal is to identify injuries to the bony spine and prevent spinal cord injury, or to stabilise and limit progression of existing partial cord injuries.

Early

• Triple immobilisation: stable (padded) surface, head and spinal motion restriction, sandbags/blanket roll, restraints (bandages/tape) ◇
• NGT, urinary catheter
• If neurogenic shock: fluid therapy, avoid SBP < 90, keep MAP ~85 1st 7 days post injury. Consider vasopressor infusion ◈
• Prophylactic antibiotics and steroid administration NOT recommended
• Analgesia
• Abnormal imaging representing acute SCI should prompt early surgical consultation (orthopaedic, neuro- or spinal surgeon)

Ongoing

• Admit unstable cervical fractures on neck traction/halo ◇
• Admit unstable thoracolumbar fractures to surgery, on strict bed-rest
• Six people min to move (use log roll and transfer board)

- Administer pressure care, i.e. 2–3-hourly turning or lifting with aggressive skin inspection and wound care

Critical documentation

- MOI, serial neurological examinations, imaging findings
- In case of clinical spinal cord clearance, document all findings in detail

Disposition

Admit documented spine injury. Refer neurological abnormalities and identified spinal lesions to a facility with spinal neurosurgical capacity.

297 Peripheral vascular trauma

Mostly associated with penetrating limb trauma, and also occurs after certain defined patterns of blunt injury, especially: posterior knee dislocation, tibial plateau fracture, supracondylar fractures of the humerus and displaced midshaft femur fracture. The major risks of delayed diagnosis are ischaemic limb loss and compartment syndrome/reperfusion syndrome.

The first five minutes

- ABC, VS, O_2, IVF (see 📖 ATLS, p. 726)
- Catastrophic bleeding from isolated limb trauma requires emergency control of haemorrhage:
  - » Direct pressure is best
  - » Indirect pressure on proximal vasculature is next option
  - » Tourniquets: evolving role in areas with long transport times, particularly with near total amputation or critical haemorrhage
- Reduce fractures and dislocations and reassess distal perfusion – attempt anatomic alignment even in unstable fractures

History and physical examination

- Pulse deficit, audible bruit, palpable thrill, pulsatile bleed or haematoma indicate arterial bleed
- Cold, painful, pale, paraesthetic, paralytic limb
- Signs of compartment syndrome (pain on passive movement)
- History of dislocation of knee – even if relocated
- Multiple or axial (not transverse) gunshot wound

Possible causes and differential diagnosis

Arterial and/or venous injury, nerve injury; fracture; compartment syndrome.

Investigations

Clinical assessment and basic imaging.

- Imaging: Doppler pressures ◇ (ankle/brachial index); plain XR ◇ (localisation of projectiles); angiogram or CT-angiogram ◈

Management

The goal of acute management is haemorrhage control and early restoration of perfusion. Transfuse early.

- Haemorrhage control: splinting, fluid resuscitation and analgesia are essential before urgent transfer to definitive care
- Early amputation of non-viable limb may be needed to prevent systemic inflammatory sequelae ◇. Basic ‘damage control’ surgery (e.g. shunting the artery) may be performed prior to transfer for definitive care
- Early fasciotomy ◇ if indicated, and transfer for definitive arterial repair or venous ligation/repair.

- Revascularisation and reperfusion injury may cause acute kidney injury
- Monitoring of serial CK essential ◈

Critical documentation

Serial neurovascular exam. Time of injury and transfer; record interventions and clinical response.

Disposition

Admit to surgical or vascular surgical unit.

298 Blast injuries

Blast injuries are often missed because the evidence of injury may be subtle and serious injury may occur in the absence of external signs of trauma. Explosions in confined areas (such as buildings) are associated with more injuries and contamination from chemicals and radioactive materials can cause additional morbidity. Blast wounds are often contaminated and may require delayed primary closure. All blast injured patients should be asked about tetanus immunisation status and given tetanus toxoid ± immunoglobulin.

Primary

Injury from the blast wave.

Lung

- Signs: cough, haemoptysis, tachypnoea, hypoxia, chest pain
- Onset of symptoms may be delayed by hours
- CXR: ‘butterfly’ pattern of bilateral patchy infiltrates, careful evaluation for PTX
- Signs of air emboli: stroke, myocardial infarction, acute abdomen, blindness, deafness, spinal cord injury, claudication
- Facemask O_2, ABC, intubation for severe hypoxaemia
- ABG initially and repeat as needed

Abdominal

- Injury occurs more in gas filled structures at the fluid/gas/solid interface e.g. colon/small bowel
- Suspect in anyone with abdominal pain, nausea, vomiting, hematemesis, rectal pain, tenesmus, unexplained hypovolaemia
- Evaluate with IDPL or FAST ◇, or CT scan ◈

Head, ears, nose and throat trauma (HEENT)

- Concussive syndrome without evidence of external trauma
- Tympanic membrane rupture is the most common primary blast injury; signs include hearing loss, tinnitus, pain, vertigo, bleeding from canal, otorrhoea

Secondary

Injury from flying bomb fragments and debris.

Eye

See 🕮 Approach to eye trauma, p. 764.

- Trauma to the eye is very common. Injuries and foreign bodies may be difficult to detect (ophthalmology consult and consider CT for diagnosis if available ◈)
- Assume a globe injury until proven otherwise
- Corneal or scleral injuries are common

• Eyelid lacerations can be extensive
• Administer broad spectrum penicillin IV if eye injury diagnosed

Tertiary

Injury resulting from being thrown by the blast wave.

Open and closed head injuries

Very common (See Head injuries, p. 755).

Traumatic amputations

• Haemorrhage control essential (correct coagulopathy with blood products)
• Broad spectrum penicillin or first generation cephalosporin
• Ensure thorough wound lavage; surgical debridement often required
• Strong association with other system injuries (chest, abdomen)

Quaternary

All other injuries, including crush, burns and exposure to toxins.

Crush syndrome

Evaluate for rhabdomyolysis when crush injury or prolonged entrapment (see Crush syndrome, p. 774).

Compartment syndrome

Suspect with prolonged entrapment or vascular injury.

Burns

See Approach to acute burn injury, p. 776.

Other

Evaluate scene and survivors for toxic chemicals/radioactive material, including inhaled toxins (CO, CN, MetHgb). Consider need for decontamination.

Disposition

• Per specific injuries
• Close follow-up of wounds, head injury, eye and ear related complaints

299 Crush syndrome

Traumatic rhabdomyolysis is the systemic release of by-products (myoglobin) of muscle injury, and is common in patients trapped in collapsed structures or otherwise immobile for extended periods. Muscle damage can result from direct trauma, toxins, ischaemia and reperfusion injury, and may be progressive with oedema. Precipitation of myoglobin in the renal tubules can cause life-threatening renal failure (acute kidney injury), but rhabdomyolysis is highly treatable if identified early.

The first five minutes

• ABC, O_2, IV, IVF (see ATLS, p. 726)
• Check for hyperkalaemia induced arryhthmias (bradycardia, wide-complex).

History and physical examination

Key historical features

• Mechanism and timing of event (prolonged immobility?)
• Degree of tissue damage (crush?)
• Amount of prehospital fluid given
• Urine output

Signs and symptoms

• Evidence of local crush injury – soft tissue, fractures
• Assess for associated compartment syndrome, neurovascular injury
• Decreased urine output, red-brown urine colour

Possible causes and differential diagnosis

• Non-traumatic rhabdomyolysis, ischaemic limbs, compartment syndrome
• Other causes of renal failure

Investigations

• Labs: electrolytes, calcium, and bicarbonate ◇, urinalysis (myoglobin may be dip positive for 'blood' with no RBC on microscopy, but absence of urine myoglobin does NOT rule out rhabdomyolysis, as serum clearance is rapid), urine SG and pH (to monitor hydration) ◇
• ECG ◇ (hyperkalaemia, arrhythmias)

Management

• IV fluid (normal saline, avoid K – no Ringers or Darrow's) most important in first 6–12 h post injury, around 500 ml/hour to maintain urine output 2–3 mls/kg/hour. * Continue IV fluid until myglobinuria resolves – monitor for acidosis
• Consider sodium bicarbonate infusion – one ampule 50 mEq in one litre half-normal saline – if significant acidosis – aim for urine pH > 6.5, watch for hypocalcaemia
• Cardiac monitoring ⟐ for arrhythmias, hyperkalaemia
• Treat hyperkalaemia
• Consider trial of mannitol if oligiuric. If no diuresis, stop as mannitol is nephrotoxic – no role for furosemide or other loop diuretics
• Haemodialysis ⟐ is indicated if rising CK (> 8 500), persistent acidosis, oliguria, pulmonary oedema or worsening kidney function
• Associated compartment syndrome or neurovascular injury may require surgical management: fasciotomy/surgery will release additional myoglobin

Critical documentation

• Suspected cause, initial exam, repeat exams and progression of symptoms, input and output
• Repeat serum electrolytes and urine testing
• Medications given

Disposition

• Admission usually required as IVF should be continued until acidosis and myoglobinuria resolve and serial CK stable below 5 000 U/L
• Patients may require transfer to higher level of care for dialysis or fasciotomy for associated compartment syndrome

300 Approach to acute burn injury

The first five minutes

- VS, IVF, O_2 by facemask (see 🕮 ATLS, p. 726)
- ABC, including early intubation for any airway/oral oedema, stridor, voice changes
- Escharotomy if restricted respiration, or limb with compromised perfusion
- Elevate burned limbs, remove jewellery
- Analgesia

History and physical examination

Key historical features

- Fire in closed space → higher risk of carbon monoxide (🕮 p. 702) or cyanide poisoning (🕮 p. 700)
- Electrical burn: AC or DC? (see 🕮 Electrical injuries, p. 208)
- Other toxin/gas exposures?

Signs and symptoms

Look for lip or mouth oedema, voice changes, facial burns, singed nasal hairs or eyebrows, or carbonaceous sputum. Respiratory insult may be thermal or chemical (local or systemic). Bronchial effects → atelectasis, pneumonia or adult respiratory distress syndrome (ARDS).

If altered consciousness, consider: head injury, CO poisoning, cyanide poisoning, burn shock, or hypoxia.

Expose all areas including the back. Remove all jewellery (swelling causes restricted perfusion).

Burn wound

Accurate assessment of total body surface area (TBSA) is essential:

- Rule of nines (Figure 300.1)
- Use patient hand (palm flat, fingers together) area as ~1% TBSA to estimate smaller areas

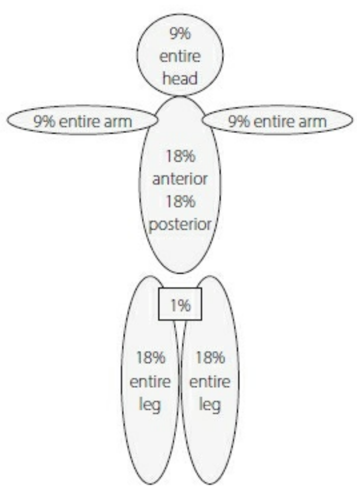

Figure 300.1 Rule of nines

Superficial wounds:

- Heal with topical management
- Superficial: pink, skin intact (not included in TBSA determinations)
- Superficial partial thickness: pink, shiny/moist, brisk capillary refill on blanching

Deep partial and full thickness:
• Require debridement and skin cover
• Deep partial thickness: red or pale, slow capillary refill on blanching
• Full thickness: may be pale/mottled or black, leathery feel, no refill on blanching

Possible causes and differential diagnosis

• Thermal (wet or dry)
• Chemical (acid or alkali)
• Electrical (AC or DC)
• Always consider hypovolaemia from other injuries

Investigations

• Labs: ABG ◇, CO level ◈
• CXR ◇

Management

The goal of acute management is to stop the burning process, anticipate complications of oedema (maintain airway), and compensate for volume loss.

Resuscitation is associated with massive fluid shifts and systemic inflammatory response with endothelial permeability.

• Facemask O_2 and early intubation for signs of airway involvement
• IV crystalloid (lactated Ringers if available): 4 ml/kg/%TBSA in first 24 h
  » Half in the first 8 h from time of burn, half in remaining 16 h
  » Add dextrose-containing maintenance fluid in children: 4 ml/kg for first 10 kg, 2 ml/kg for next 10 kg and 1 ml/kg > 20 kg
  » Titrate to patient response: target 0.5–1 ml/kg/hour urine output
  » Caution: airway oedema may progress with resuscitation
• Bladder catheter to monitor output

Escharotomy

Burns coagulate the skin to form rigid eschar.

Escharotomy is indicated in circumferential deep partial or full thickness burns to the torso (with respiratory compromise or abdominal compartment syndrome) or a limb (compartment syndrome) (see 🕮 Escharotomy, p. 856).

Wound management

As per local burn centre instructions, or gently clean with antiseptic and debride blisters. For transfer: sterile clingfilm/plastic wrap held in place with bandages. Add topical antimicrobial dressings based on availability (chlorhexidine/silver sulphadiazine/moist nanocrystalline silver dressings).

Critical documentation

• Airway exam and interventions
• Size, depth and location of burns
• Serial exams and response to interventions (airway or escharotomy)
• Mechanism of burn and comorbidities

Disposition

Refer to regional burn centre for:
• Associated inhalation or polytrauma injury
• Partial thickness burns > 10% TBSA in children, > 15% TBSA in adults

- Full thickness burns > 5% TBSA
- Face, hands, major joints, feet, genitals, and all circumferential burns
- Electrical and chemical burns
- All children < 12 months and the elderly (see Domestic and intimate violence victims, p. 907)
- Major comorbidities or pregnancy

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Part

5

Emergency ultrasound

301 Introduction to ultrasound

US is best learned by doing, and this section is not intended as a source of primary training, but as a rapid reminder of a few indications and views for those who have already received some training. US is a dynamic study, and video images communicate much better than the words and static images we can present here. Where access is available, we encourage use of the excellent free online resources listed in the references on p. 802.

The goal of a rapid bedside US study is usually to answer a specific clinical question, to gather information that will aid in diagnosis or in a procedure. To use US effectively, you must know the diagnostic test performance for each indication: how good (how sensitive and specific) is US for the question you are asking?

As for many diagnostic tests, including the physical exam, the performance and interpretation of US is experience-dependent. US findings should be synthesised with other clinical information, with an honest evaluation of your confidence in performing the study, just as you might decide how much weight to give your stethoscope evaluation of heart sounds. Never let a normal US override a high level of clinical concern for a dangerous diagnosis. The patient who is hypotensive and complaining of abdominal pain after an MVC needs further evaluation and intervention no matter what the US shows, though US may help guide priorities.

The impact of US point of care (POCUS) on management is highly context-dependent. The management implications of a positive FAST in an initially stable trauma patient, for example, may vary depending on whether there is CT available at the facility and whether observation and non-operative management are available for solid-organ injury.

The basic US movements mentioned in this section are shown below. No single US image will give you the information you need. Learn the basic movements and use them in combination to thoroughly ‘interrogate’ the structure of interest. In particular, make sure you have performed all of the components of a given US study before deciding that it is negative. Knowing the components and constraints of a given study is essential to high-quality US and is beyond the scope of this brief overview section. We hope those who currently use US will find these chapters a useful review, and that those who are new to US will find in them inspiration to seek training and integrate US into their clinical practice.

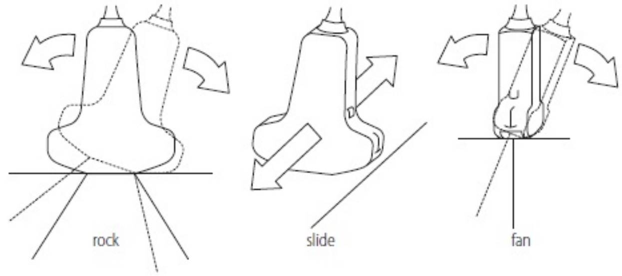

Figure 301.1

For those interested in setting up a POCUS training program, the International Federation for Emergency Medicine recommends the following components.

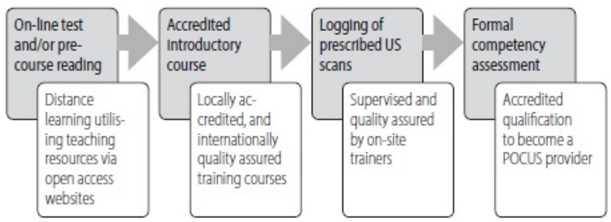

Figure 301.2 POCUS training programme components as recommended by IFEM

302 Ultrasound for trauma

Focused assessment with sonography in trauma (FAST)

The FAST is a limited US study with one goal – to identify free fluid in dependent areas of the supine patient that, in the setting of trauma, is presumed to be blood. The associated clinical question is whether visualised fluid is the cause of haemodynamic compromise. FAST is specific, but not perfectly sensitive – it should NEVER be used to ‘rule-out’ injury, but free fluid that is visualised on US does correlate well with operative findings. Most importantly, a negative FAST should never override high clinical suspicion of injury. FAST is most helpful in hypotensive patients to identify whether bleeding into the abdomen, chest, or the pericardium is responsible for the haemodynamic compromise. The basic FAST examines the hepato-renal, spleno-renal, sub-phrenic, and retro-vesicular pelvic regions in addition to the pericardium. An extended version, or E-FAST, includes evaluation of the chest for evidence of PTX and HTX.

Perform E-FAST during (if two providers are available) or just after the primary survey and resuscitation phase, using a large curvilinear or a phased array probe with the patient supine. Slight Trendelenburg position increases sensitivity of upper abdominal views. See image section for the views described below.

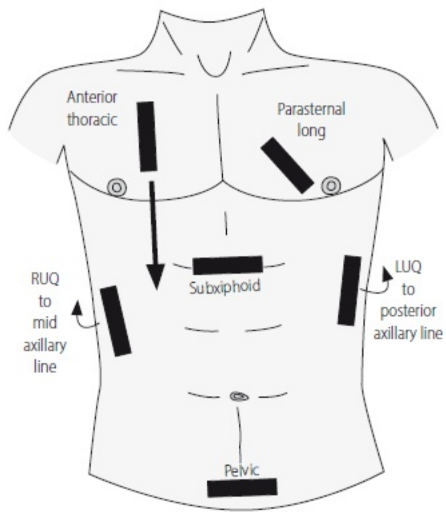

Figure 302.1 E-FAST probe positions

a. RUQ view

- Place probe in mid-axillary line, 8–11th intercostal space, with the probe marker towards the head, and fan slightly posterior (lift the hand so the probe points slightly towards the bed). Morrison's pouch is the emphasis of this view, but free fluid may appear anywhere along the edge or tip of the liver, so systematically 'interrogate' the entire area, including the sub-phrenic region. Minimse rib shadows by rotating the probe counter-clockwise (posterior) to view between the ribs. (See image section for FAST.)
- Slide cephalad and/or rock superiorly to view the diaphragm and look for pleural fluid (black stripe or triangle) just superior to the diaphragm. (📖 US for dyspnoea, p. 787)

b. Cardiac views

- **Subxiphoid:** place the probe in the subxiphoid region, 2 cm below and to the right of the xiphoid in a flat 10° angle to chest wall with the probe marker to the patient's right (see image section). Adjust the depth to evaluate the entire pericardium. Aim the probe slightly towards the patient's left as needed to visualise the heart. A dependent pericardial effusion will appear near the top of the screen between the liver and the heart.
- **Parasternal long-axis:** place the probe just left of the sternum, 3rd/4th intercostal space, directly over the centre of the heart. Reduce the depth. Pericardial effusions track anterior to the descending thoracic aorta; left pleural effusions track posteriorly. Slide the probe toward the left hip to view the apex, where pericardial effusion is usually visible.
- ANY amount of pericardial effusion is significant in the setting of trauma, as it indicates injury to major thoracic structures.

c. LUQ view

- Place the probe in the posterior-axillary line, 6–9th intercostal space, with the probe marker towards the head (see image section). Slide the probe cephalad, fan slightly posterior to view the lower tip and superior surface of the spleen. Rotate slightly clockwise (posterior) as needed to view between the ribs.
- Move the probe 1–3 rib spaces fanning and rocking the probe in each rib space through all planes to visualise the inferior pole of the left kidney and the superior extent of the left paracolic gutter.
- Slide the probe cephalad to look for pleural fluid just superior to the diaphragm.

d. Pelvic view

- Place the probe just cephalad to the pubic bone in the midline with the probe marker cephalad to produce a longitudinal view (see image section). A full bladder is triangular and the lower angle divides the peritoneum (left side of screen) from the pelvis. In males, free fluid will collect just posterior to the bladder. In females, free

fluid will be seen just posterior to the uterus (pouch of Douglas) and may also surround the edges of the uterus.

- Rotate the probe 90°, with the marker to the patient's right for transverse views.

e. Anterior thoracic views

- Place a high-frequency linear array transducer longitudinally with the probe marker towards the head. Start in the 2nd intercostal space in the mid-clavicular line. Adjust the depth to centre the pleural line (usual maximum depth is 4 cm). Adjust the probe until one rib is visible on each side of the image and the intra-costal space is centred. Anchor the probe and look for the sliding motion of the pleura ('marching ants') at the posterior border of the ribs and for the presence vertical echoic lines ('comet-tails') extending from the pleural line. These are normal findings. The absence of pleural sliding strongly suggests PTX. Continue the study in multiple rib interspaces, sliding from the 2nd through the 5th intercostal spaces at the mid-clavicular line. Sensitivity is increased by also scanning through the anterior and mid-axillary lines. See www.sonoguide.com for video images of lung sliding and comet tails.
- Switch to M-mode. A normal lung will produce the linear appearance of the more static chest wall tissues ('waves') above the pleura, over the granular appearance created by pleural sliding below ('sandy beach') – 'waves on the beach' is the normal finding. In PTX, the linear hyperechoic lines extend thorough below the unmoving pleura. Always compare one side of the chest to the other. See image section.

Use of ultrasound during resuscitation

Determine the intravascular volume status by looking at the collapsibility of the IVC in the subxiphoid view (📖 Cardiovascular ultrasound, p. 790). An IVC collapse > 50–60% with inspiration suggests hypovolaemia. Track changes with volume resuscitation.

Use of ultrasound in the secondary survey

A high-frequency linear array transducer may be used to evaluate for fractures which appear as a discontinuity of the bright bony cortex. Associated soft tissue haematoma may also be visible.

Repeat the complete EFAST after an interval of 30 minutes.

303 Ultrasound for dyspnoea

Dyspnoea may result from a range of abnormalities in multiple organ systems (see 📖 p. 630 for a system-based differential). Combining limited pulmonary, cardiovascular, and abdominal US studies allows rapid evaluation of cardio-pulmonary physiology and can identify a range of possible aetiologies in the patient with undifferentiated dyspnoea. Techniques for these studies are described in other chapters in this book, at www.sonoguide.com and in the open-access *Partners in health manual of ultrasound for resource limited settings* (📖 p. 802).

Basic principles of lung ultrasound

Air and fluid

Air appears bright or *echogenic* on US, and fluid dark, or *hypoechoic*. A normal air-filled lung has an indistinct hazy appearance, while a pleural effusion will usually appear as a black area with distinct borders. A fluid-filled segment of lung, or consolidation, will appear somewhere in between, most similar to the US density of liver, and may be punctuated with bright white areas representing *air-bronchograms*, or airway segments visibly distinct from surrounding fluid-filled lung. See image section for a figure of air-fluid-consolidation.

Remember that air in the body goes up and fluid down – this determines where we look in the supine patient for pulmonary pathology, such as PTX (anterior chest) or effusion (posterior thoracic cavity).

Artefacts

Much of lung US involves looking at *artefacts* – these are not actual structures, but image patterns produced by the interaction of fluid and air in the lung. **A-lines** are produced by reverberation and appear as a series of horizontal

lines at regular intervals. They are seen in normal lung and remain visible in PTX. **B-lines** arise from the pleural line and extend to bottom of screen. They move with the lung and efface A-lines. More than 2 B-lines in a single rib space represents pulmonary oedema.

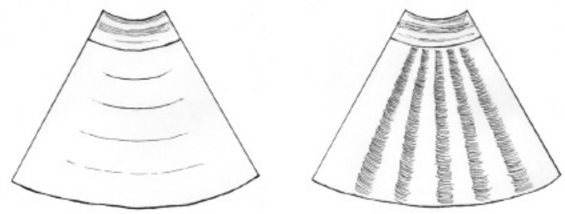

Figure 303.1 A-lines (left) and B-lines (right)

Differential diagnosis

In the trauma patient presenting with dyspnoea, thoracic US is usually focused on evaluation for PTX and HTX, but may also be used to evaluate for rib or sternal fractures. In the medical patient presenting with dyspnoea, US can be used to evaluate for consolidation, pulmonary oedema, and of course, PTX, especially valuable in patients on positive pressure ventilation. A normal lung US in the dyspnoeic patient increases the likelihood of diagnoses such as bronchospasm and PE, or of non-pulmonary diagnoses, such as anaemia or acidosis.

Specific studies

Pneumothorax

US has very good sensitivity and specificity for PTX in the supine patient. See 🕮 p. 786 for technique.

Pulmonary oedema

Pulmonary oedema is increased extravascular lung water (due to dysfunction of the alveolar-capillary membrane) and appears as B-lines on US. One to two isolated B-lines, especially in the lateral lung fields may be normal, but more indicate oedema. There are specific criteria for the number of B-lines that suggest interstitial versus alveolar oedema, but the important principle is that *the number and crowding of the B-lines increases with greater loss of aeration*. Pulmonary oedema associated with CCF is usually bilateral. Unilateral oedema suggests other diagnoses, such as pneumonia with oedema, or foreign body with lobar collapse. See Consolidation below.

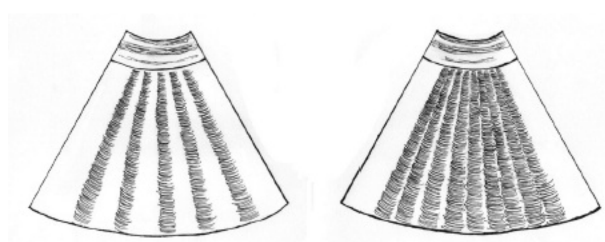

Figure 303.2 Increasing B-line density

Effusion

Start from the right or left upper quadrant FAST view and slide and/or rock superiorly to view the diaphragm; look for pleural fluid (black stripe or triangle) just superior to the diaphragm. See image section. In normal patients, the costophrenic angle is not clearly visualised because it is filled with aerated lung. Seeing distinct anterior and posterior edges of the costophrenic angle on US suggests the presence of fluid.

Consolidation

Fluid filled lung has a similar appearance to liver on US, so ensure that the visualised structure is in the thoracic cavity. Consolidation should arise from the pleural line (or the edge of a pleural effusion). There may be a focal area of B-lines nearby, as consolidation often has associated oedema. As the patient breathes, caudal inspiratory movement may be present as the diaphragm moves down, but there should be minimal anterior-posterior shift as there is no air filling of the consolidated area.

Cardiovascular

Cardiac US can provide evidence of several diagnoses that may present with dyspnoea, including cardiac failure, pericardial effusion, and tamponade. TB pericardial effusion, in particular, is often characterised by a distinct appearance with fibrinous material that can be seen waving within the effusion. Right heart strain may suggest PE, and a normal pulmonary US combined with a positive DVT study has high specificity for PE.

Other causes

US may also be used to diagnose non-cardiopulmonary conditions that cause dyspnoea, including large-volume ascites that may limit inspiration, and poor diaphragmatic excursion associated with musculo-skeletal weakness and neurologic conditions.

304 Cardiovascular ultrasound

Basic cardiac US can be used to rapidly evaluate for pericardial effusion, tamponade physiology, chamber size and gross estimate of LV function.

Indications

Common indications for bedside cardiac US: hypotension, chest pain, cardiac arrest, dyspnoea, lower limb oedema, sepsis, murmurs, muffled heart sounds.

Cardiac views

There are four basic views (see Figures 304.2–304.5):
• Parasternal long axis
• Parasternal short axis
• Apical four chamber view
• Subxiphoid view

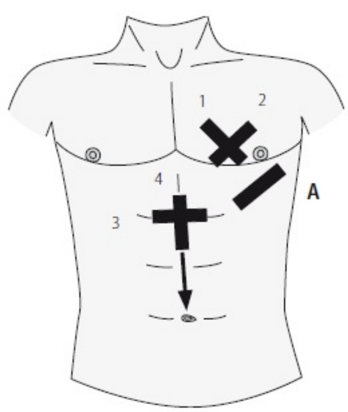

Figure 304.1 Thorax with probe positions

Parasternal long axis (probe position 1)

Technique

Place patient supine or onto their left side. Orient probe to left of sternum (3rd–5th rib space), with marker pointing towards patient’s right shoulder.

Moving down the screen from the top the structures seen are: a portion of the right ventricle and outflow tract (RVOT), LV and outflow tract, aorta, LA and mitral valve.

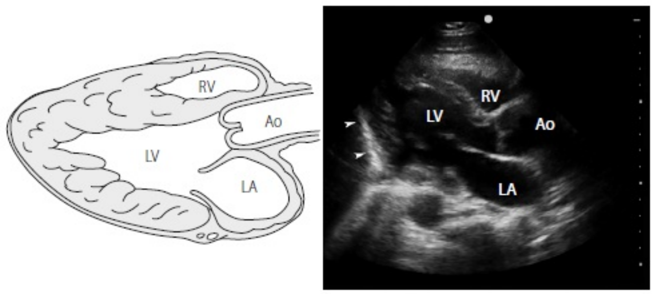

Figure 304.2 Parasternal long axis view

Source: Used with permission from Partners in Health Manual of Ultrasound for Resource Limited Settings

Clinical decision making

In a normal heart, RV, aortic root, and LA appear will be about the same size. Relative enlargement of any of these structures suggests dilatation. Parasternal long view is useful for detecting pericardial effusion/tamponade, and mitral stenosis (usually a result of rheumatic heart disease in Africa). Stenotic valves may appear thick walled and calcified with poor opening. In a normal mitral valve, anterior leaflet should approach the intraventricular septum.

Parasternal short axis view (probe position 2)

Technique

From the parasternal long axis, turn the probe clockwise 90° so its marker points near the patient’s left shoulder (2 o’clock). This view makes the left ventricle look like a circle and the right ventricle appears as a half circle attached to side.

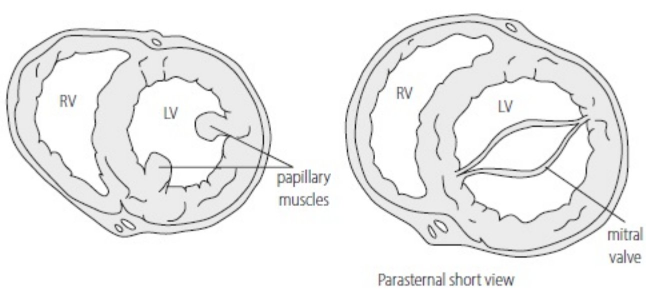

Figure 304.3 Parasternal short axis view

Source: Used with permission from Partners in Health Manual of Ultrasound for Resource Limited Settings

Clinical decision making

Parasternal short axis view is very useful in estimating LV function and ejection fraction.

Apical four chamber view (probe position A)

This view allows relative comparison of chamber sizes.

Technique

Place the probe at the apex of the heart (at the nipple level in the anterior axillary line, around the 4th–5th rib space).

The LV will appear on the right side of the screen, and the RV on the left side.

Figure 304.4 Left: photo position of the probe Right: US image (apical four chamber view)

Source: Used with permission from Partners in Health Manual of Ultrasound for Resource Limited Settings

Clinical decision making

This view is good for assessing relative chamber size and identifying atrial enlargement and RV dilatation (associated with a small and compressed left ventricle).

Subxiphoid view (probe position 3)

Place probe on subxiphoid region with marker towards patient's right and aim probe towards head. Try increasing pressure and flattening angle of the probe as needed to improve view. Bent knees will help relax abdominal muscles.

This view will display ventricles at an angle, with RV anterior, RA (left screen) and LV (right screen) below that, and LA at the bottom of the screen.

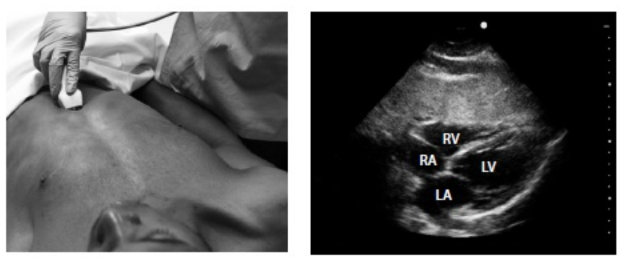

Figure 304.5 Left: position of the probe Right: US image (subxiphoid view)

Source: Used with permission from Partners in Health Manual of Ultrasound for Resource Limited Settings

IVC (probe position 4)

The IVC is a compliant structure and its diameter depends on a variety of factors, including intravascular volume status and intrathoracic pressures. US measurement of IVC diameter, and specifically, measurement of degree of collapse (decrease in diameter) during inspiration can provide an estimate of volume status.

Technique

Place the patient in supine a position and use a curvilinear probe. Position the probe (with the marker towards the head) in a longitudinal orientation in the sub-xiphoid area (position 4 in Figure 304.1, sliding or fanning 1–2 cm to the patient's right as needed to get a clear image). The IVC diameter should be measured 2–3 cm from the right atrial border, during inspiration and expiration.

Figure 304.6 US image of IVC at R atrium (IVC1); IVC inspiration; IVC expiration
Source: Used with permission from Partners in Health Manual of Ultrasound for Resource Limited Settings

Clinical decision making

The per cent collapse of the IVC with respiratory variation should be calculated, and collapse > 50–60% suggests intravascular hypovolaemia. Collapse < 50% (which loosely correlates with a CVP > 8–10 mmHg) suggests that hypovolaemia is unlikely to be the cause of hypotension. Note that in intubated patients, respiratory dynamics of IVC diameter will be reversed: minimum IVC diameter is seen on expiration and maximum on inspiration. In either case, the fractional collapse is calculated as the difference between maximum and minimum diameters, divided by the maximum and multiplied by 100 to get the per cent.

Aorta (probe positions 3 and 4)

US is very useful in the diagnosis of aortic aneurysms, and may show (but is a poor study for) aortic dissection. US is not a sensitive study for evaluating aneurysmal leak or rupture, but in a patient presenting to an acute care setting with abdominal or back pain, hypotension, or syncope, identifying the presence of an aneurysm can greatly expedite provisional diagnosis and management. Identifying a dissection on US can be extremely helpful, but a large proportion of dissections will not be seen on bedside US, and CT angiography is the study of choice.

Technique

Place the patient supine and use a curvilinear/phased array probe. Adjust the depth as needed as the aorta is very posterior. Starting from the subxiphoid space (position 3), with the probe marker to the patient's right, apply steady gentle pressure to displace bowel gas. Identify the spine shadow. The aorta is just superficial and to the right of the vertebral bodies, and appears as a thick-walled, round, fluid filled structure in cross-section. The IVC is a thin walled collapsible just to the right (screen left) of the aorta and the spine. It usually appears ovoid, but may be flat and difficult to visualise in dehydrated patients. Freeze and measure the maximum diameter of the aorta (including the wall) by using callipers. Normal size is less than 3 cm. Rotate the probe marker towards the patient's head to get a longitudinal view (position 4) and measure the maximum diameter in this view as well. A complete study includes a continuous scan of the aorta (without lifting the probe) in both planes (slide down from positions 3 and 4 in Figure 304.1). Note that a complete study requires a scan from the xiphoid process (visualising the celiac trunk) to the umbilicus (fanning to view the iliac bifurcation). Measure diameter with callipers in at least three locations in each orientation.

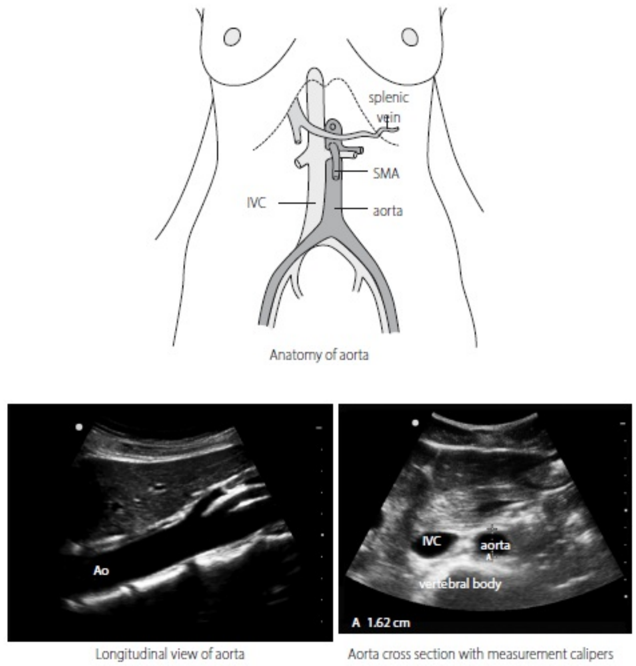

Figure 304.6 Anatomy of aorta; longitudinal section of aorta; image of aneurysm.

Source: Used with permission from Partners in Health Manual of Ultrasound for Resource Limited Settings

Clinical decision making

The definition of aneurysm is an aortic diameter of > 3 cm in any view. An AAA greater than 5 cm has a high risk of rupture, and an aneurysm of any size in a symptomatic patient requires further evaluation (p. 112).

Aortic dissection can be detected in a longitudinal or transverse view, and can be seen as hyperechoic intimal flap, appearing as a bright white line within the vessel. Thrombus may also be directly visible.

If aneurysm or dissection is seen, attempt to identify the level and extent (level of the start and end point based on surrounding structures).

305 Ultrasound-guided vascular access

The aim of this chapter is to describe the technique of US-guided cannulation of peripheral and central veins.

Technique

For proper orientation, the operator should stand opposite to the screen and arrange all tools needed in advance, including probe cover, gel, gloves, tourniquet, skin cleanser, and all IV placement supplies. A linear probe should be used with the marker facing the same direction as the screen indicator.

Distinguish veins from arteries by their compressibility, thin walls, lack of pulsations, and ovoid shape. Colour doppler can show pulsatile blood flow that may help distinguish arteries from veins, but the colour of flow only indicates whether flow is towards or away from the probe. There are two techniques for vessel puncture as illustrated in Figure 305.1.

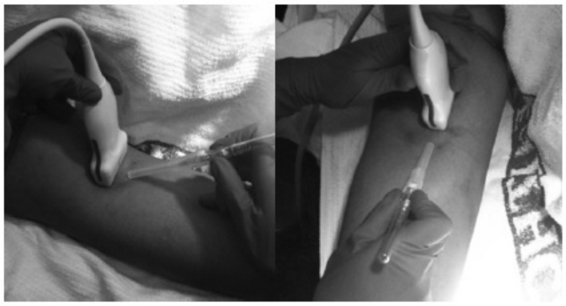

Figure 305.1 Left: out of plane or cross sectional technique Right: in plane technique

Visualise the target vein in the long (in plane technique) or short axis (out of plane or cross sectional technique) (Figure 305.2).

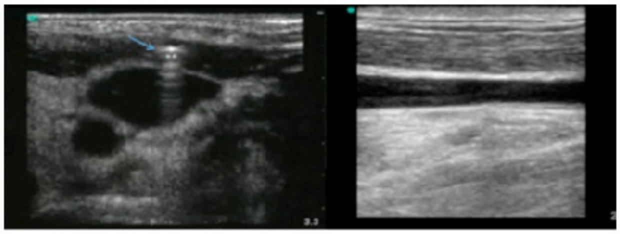

Figure 305.2 Left: out of plane technique. Arrow shows hyperechoic needle tip ('ring-down' artefact) approaching vessel Right: in plane (long axis) view of vessel

Note the depth of the vessel using the centimetre guide on the right side of the screen. Insert the needle the same distance from the probe at a 45° angle (see Figure 305.3). Follow the bright (hyper-echoic) needle tip progression through the grey subcutaneous tissue. When close to the vessel, look for a blood 'flash' in the catheter, advance the catheter as usual.

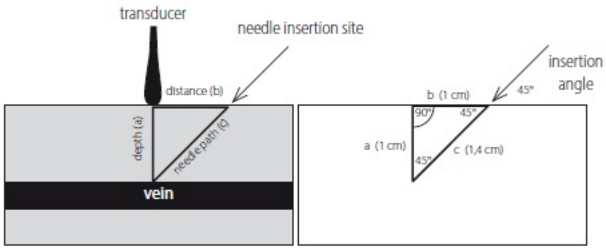

Figure 305.3 45° angle

Source: Sonoguide.com, used with permission

Safety

If arterial puncture occurs, hold pressure until bleeding resolves. Avoid infection by thorough cleansing of the skin prior to puncture and by using a sterile glove or cover on the probe. Central access must be done in a completely sterile fashion, preferably with cardiac monitoring.

306 Ultrasound for hypotension

Combining limited cardiac, abdominal, pulmonary, and vascular US studies can allow rapid evaluation of cardiopulmonary physiology and identify a range of possible aetiologies in the patient with undifferentiated hypotension. Techniques for these studies are described in other chapters in this book, at www.sonoguide.com and in the open-access *Partners in Health Manual of Ultrasound for Resource Limited Settings* (📖 p. 802).

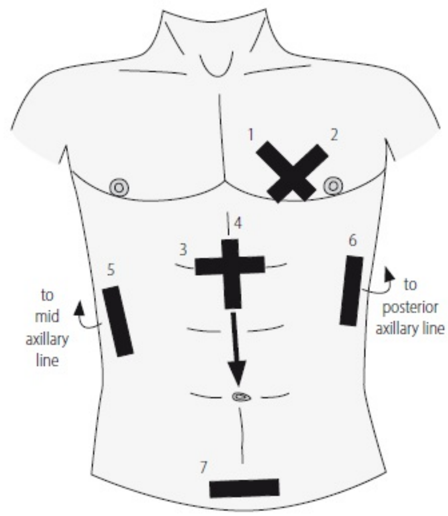

Figure 306.1 Thorax with probe placements

Cardiac: parasternal long (1) and short (2) axes

Evaluate for pericardial effusion and tamponade, RV strain (suggestive of pulmonary outflow tract obstruction, as in pulmonary embolism) and assessment of LV contractility (visual estimation based change from diastole to systole). Poor cardiac contractility may result in decreased cardiac output and hypotension. (📖 p. 798)

Abdominal ultrasound

Evaluate for free fluid (see US in Trauma chapter for technique, position 5, 6, 7 in Figure 306.1) and aortic aneurysm (sliding down from positions 3 and 4 in Figure 306.1). In combination with other clinical signs and symptoms, this may identify potential causes of hypotension, including AAA, internal blood loss, ruptured ectopic pregnancy, or ruptured viscus.

Inferior vena cava (IVC)

US measurement of the IVC provides a non-invasive estimate of the intravascular volume status of the hypotensive patient. The technique is described on 📖 p. 792 and the view is obtained from probe positions 3 and 4 (fan to patient's right) above.

Pulmonary ultrasound

Pulmonary US can identify PTX and HTX, both of which may cause hypotension. In addition, US may provide evidence of pulmonary oedema, suggesting cardiac failure and poor cardiac output, or consolidation, which may be associated with hypoxia and septic shock. These techniques are described in 📖 p.784 and p. 787 .

307 Ultrasound to expedite the diagnosis of TB in HIV patients

HIV has changed the pathology, histology, clinical presentation and epidemiology of TB. Nearly 25% of newly diagnosed TB patients who are co-infected with HIV present with extra pulmonary TB (EPTB), which may be missed by current sputum-based testing strategies. The Focused Assessement with Sonography for HIV-associated TB (FASH) can be used to rapidly identify signs that are highly suggestive of EPTB in high-prevalence settings.

Position	Positive findings	Exam
RUQ	1. Pleural effusion 2. Free fluid	Begin as if doing a RUQ FAST. Interrogate Morrison's Pouch for free fluid, and slide caudally to evaluate for pleural effusion. Then interrogate the entire parenchyma of the liver, looking for hypoechoic focal liver lesions.

	3. Liver lesions	
LUQ	4. Pleural effusion 5. Free fluid 6. Splenic lesions	Start as if doing a FAST in the LUQ. Interrogate the splenorenal space for free fluid, and slide caudally to evaluate for pleural effusion. Then interrogate the entire spleen, looking for hypoechoic lesions measuring 0.5–2.0 cm.
Subxiphoid	7. Pericardial effusion 8. Abdominal lymph nodes > 1.5 cm	Start with the probe just inferior to the xiphoid, as in the cardiac subxiphoid view. Evaluate for pericardial fluid. Tilt the probe up so that it is nearly perpendicular to the patient's skin, with the marker pointed to the patient's right. In this position the periportal area can be visualised. Slowly move the transducer caudally to assess the periaortic area. Interrogate the area for lymph nodes > 1.5 cm.
Suprapubic	9. Free fluid	Start with the probe just caudal to the pubic bone in either the transverse or sagittal orientation. Interrogate the area for free fluid as you would in a FAST.

Integrating the FASH study

The FASH is designed to be used in HIV+ patients for whom there is a high clinical suspicion for TB. The following algorithm describes the integration of FASH into a general diagnostic pathway for these patients:

Test for TB with two sputum smears for improved yield
If smears are negative or unavailable, proceed with the following:

- Sputum culture – × 2
- History and exam: initiate treatment if any clinical evidence for TB
- CXR: initiate treatment if any radiological evidence for TB
- FASH US scan: initiate treatment if any evidence for EPTB

Validating the FASH scan results in your own setting

- When used in the population described above, the sensitivity and specificity of FASH for detecting EPTB is 95% and 98% respectively
- Remember, **in high prevalence settings** the positive predictive value (PPV) of FASH for TB will be high and the negative predictive value (NPV) will be low
- **In low prevalence settings**, the PPV of FASH for TB will be low and NPV high, which undermines the FASH scan's utility as a diagnostic test

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Part

6

Procedures

308 Procedural analgesia and sedation

ACEP defines procedural sedation as a technique of administering sedatives or dissociative agents with or without analgesics to induce a state that allows patient to tolerate unpleasant procedures while maintaining cardiorespiratory function. Procedural sedation and analgesia (PSA) is intended to result in a depressed level of consciousness that allows patient to maintain oxygenation and airway control independently:

- Minimal sedation: the patient responds normally to voice
- Moderate sedation: the patient responds purposefully to voice alone or light tactile stimulation. the goal for most procedures
- Deep sedation and analgesia: the patient responds to painful stimulation

In GA the patient cannot be aroused, requires airway protection and ventilation support. Dissociative sedation is a

trance-like cataleptic state with profound analgesia and amnesia; airway reflexes are maintained.

Indications and contraindications

Indications

Painful procedure which would cause unacceptable patient distress, or in which a calm and cooperative patient is essential but where GA is not possible or desirable.

Contraindications

- If required level of sedation cannot be provided safely, consider alternatives: regional anaesthesia, GA (by anaesthetist), or transfer to facility with a higher level of care
- Two providers must be available: person who supervises sedation should not perform any other function, and must have skills and equipment to rescue from inadvertent GA

Preparation

1. Focused history: comorbidities, prior adverse with sedation/anaesthesia.
2. Fasting: ideally for six hours prior to sedation. However, urgent procedures may require that sedation be performed earlier
3. Perform an airway examination and a cardiac/lung examination. If a difficult airway is anticipated, consider alternatives
4. Equipment is available and functioning
5. Consider length of procedure and desired depth of sedation. Select medication (Table 308.1) and calculate dose required to induce and maintain sedation:
   - Ketamine's unique dissociative state: makes it a good choice for resource-limited settings, as non-physicians can safely administer it
   - Etomidate has minimal cardiopulmonary effects
   - Propofol causes some muscle relaxation, which can be useful in orthopaedic procedures

   Remember that adequate analgesia is required: use IV morphine or fentanyl.

Table 308.1 Common sedatives used for procedural sedation

Medication	Dose (IV)	Onset	Duration
Etomidate	0.1–0.2 mg/kg	<1 min	3–5 min
Propofol	1 mg/kg	<1 min	3–10 min
Ketamine	1 mg/kg (5 mg/kg IM)	5 min	15–30 min

Consent

Risks include hypoxia, vomiting, aspiration, hypotension, inadequate sedation or analgesia, and cardiac arrest; ketamine causes re-emergence phenomena. Benefits include facilitation of the procedure. Alternatives include regional block, systemic analgesia or admission for general anaesthetic.

Equipment

- Instruments for continuous pulse oximetry and BP measurements
- Continuous cardiac monitoring ◇
- Supplemental O_2
- Capnography ◈
- Resuscitation equipment
- Reversal agents, e.g. naloxone and flumazenil

Procedure

1. Administer the medication.
2. Announce once the desired depth of sedation has been achieved.
3. Continue to monitor the patient for adverse effects and depth of sedation. Re-dose as necessary.

Aftercare

Continue monitoring once primary procedure is completed: without painful stimulation, patient is now at risk for complications associated with deep sedation. Observe until full recovery (normal VS, alert, following commands).

Potential complications

See consent (above).

Documentation

Document indication, NPO status, medications, patient response, adverse events, aftercare.

Disposition

Admit as needed for underlying condition. If discharged, leave with a reliable adult who can observe and assist. Patients cannot drive, operate machinery, make important decisions or consume alcohol or drugs for 12 hours.

309 Local anaesthetic nerve blocks

Indications and contraindications

Indications

Pain relief and facilitation of painful procedures.

Contraindications

Any infection overlying injection site. Bleeding disorder. Allergy to local anaesthetic. Compromised distal circulation (digits).

Preparation

Consent

Risks include infection, injection into vessel, compartment syndrome, haematoma, and nerve damage. **Benefits** include facilitation of therapeutic procedure. **Alternatives** include procedural sedation and admission for GA.

Equipment

- Skin antiseptic, sterile guaze, clean gloves
- 10 ml sterile syringe, 25 gauge sterile needle
- Local anaesthetic with or without adrenaline

Dental block

- Topical anaesthetic (benzocaine 20% or lignocaine 5%)
- Local anaesthetic (lignocaine 2% or bupivacaine 0.25%)
- 3 ml syringe with 25–27 gauge needle, 3–5 cm in length

Procedure

Regional nerve blocks

Always:

- Aspirate before injecting
- Inject anaesthetic slowly
- Expect effect in 2–20 minutes
- If using bupivacaine, exercise caution due to potential cardiotoxicity

Wrist

Ulnar nerve:

- Goal: anaesthetise medial dorsal/volar hand of little finger and ulnar side of ring finger and ulnar aspect of hand
- Anatomy: nerve runs medial to ulnar artery beneath flexor carpi ulnaris tendon
- Method: position patient's palm up with thumb opposing little finger. Identify the most medial tendon. Insert needle between the distal ridge of ulna and the tendon. Advance posterolaterally until you reach bone. Withdraw slightly, inject 5 ml of anaesthetic.

Median nerve:

- Goal: anaesthetise lateral dorsal/volar surface of lateral 3 ½ digits, including dorsal distal phalanges and the medial aspect of hand
- Anatomy: nerve runs medial to flexor carpi radialis tendon, lateral to the palmaris longus
- Method: position patient's palm up with hand in fist and wrist flexed. Two tendons will show. Insert the needle 1 cm proximal to the proximal wrist crease and 1 cm medial to the flexor carpi radialis. Advance 1 cm or until a paraesthesia or a pop through the retinaculum is felt. Inject 5 ml of anaesthetic.

Radial nerve:

- Goal: anaesthetise dorsal lateral of lateral 3½ digits, excluding dorsal distal phalanges/nails
- Anatomy: nerve runs lateral to radial artery
- Method: position patient's wrist in neutral position. Palpate artery at level of proximal palmer crease. Insert needle at depth of artery, just lateral to it. Inject 5 ml of anaesthetic. Withdraw needle and extend block through subcutaneous space from injection site to mid dorsum using additional 5 ml of anaesthetic

Ankle

Posterior tibial nerve:

- Goal: anaesthetise plantar surface of the foot
- Anatomy: nerve runs posterior to medial malleolus and posterior tibial artery
- Method: palpate the artery over superior/posterior aspect of medial malleolus. Insert needle posterior to the artery until you reach bone, then withdraw slightly; inject 5–10 ml of anaesthetic

Superficial peroneal and sural nerve:

- Goal: anaesthetise anterior foot/toes, lateral plantar foot
- Anatomy: nerves run superficially between lateral malleolus and Achilles tendon
- Method: draw an imaginary horizontal line from the anteriomedial rim of lateral malleolus to ipsilateral edge of Achilles. Insert the needle subcutaneously at distal aspect of lateral malleolus. Inject 10 ml of anaesthetic subcutaneously along the imaginary horizontal line

Upper leg

Femoral nerve block:

- Goal: anaesthetise the thigh (useful for femur fractures)
- Anatomy: femoral nerve found lateral to femoral artery, inferior to inguinal ligament and posterior to fascia iliaca
- Method: place patient supine with leg in neutral position. Find pulsating femoral artery just under the inguinal ligament and insert needle 2 cm lateral to it. Advance needle posteriorly until two distinct pops through fascia lata and fascia illiaca are felt. If parasthesia felt, withdraw until resolves. Hold tight pressure just distal to injection site and inject 15–20 ml anaesthetic. Continue holding pressure for 5 mins to facilitate proximal spread of anaesthetic.

Upper face

Supraorbital nerve:

• Goal: anaesthetise forehead and nasal ridge

• Anatomy: nerve runs through supraorbital foramen

• Method: identify the foramen above mid-superior orbital rim by palpating for a bony groove. Insert needle adjacent to the foramen until you reach bone, then withdraw slightly. If the patient feels parasthesia, withdraw until resolves. Inject 5 ml of anaesthetic

Infraorbital nerve:

• Goal: anaesthetise lower eyelid, upper lip, and medial cheek

• Anatomy: nerve runs through infraorbital foramen

• Method: identify the foramen below mid-inferior orbital rim by palpating for a bony groove. Anaesthetise as outlined for the supraorbitlal nerve

Digital blocks

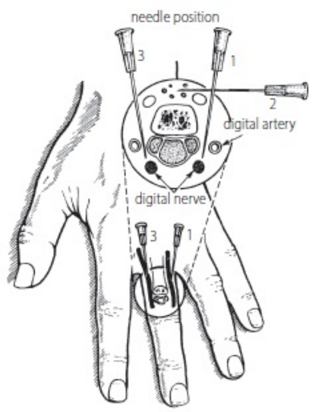


Figure 309.1 Digital block

Source: Brown & Wyatt. 2008. *Oxford American Handbook of Emergency Medicine*. By permission of Oxford University Press, USA.

• Document neurologic examination of the digit before the procedure

• Clean the digit

• Using a needle, slowly inject local anaesthetic on the dorsolateral aspect of the base of the finger or toe and raise a small skin wheal

• Direct the needle anteriorly toward the base of the phalanx. The needle is advanced until it contacts the phalanx; observe for any protrusion from the palmar dermis directly opposite the needle path

• 1 ml of solution is injected as the needle is withdrawn 1 to 2 mm from the bone contact. Additional 1 ml is injected continuously as the needle is withdrawn back to the skin

• Repeat procedure on each side of the base of the finger or toe to achieve anaesthesia of the entire digit

• Wait about 5–10 minutes for adequate anaesthesia

Dental blocks

• Apply topical anaesthesia to the mucosa site, if available

• Draw up 3 ml of anaesthetic solution into the syringe and load a 25G needle

Anatomy

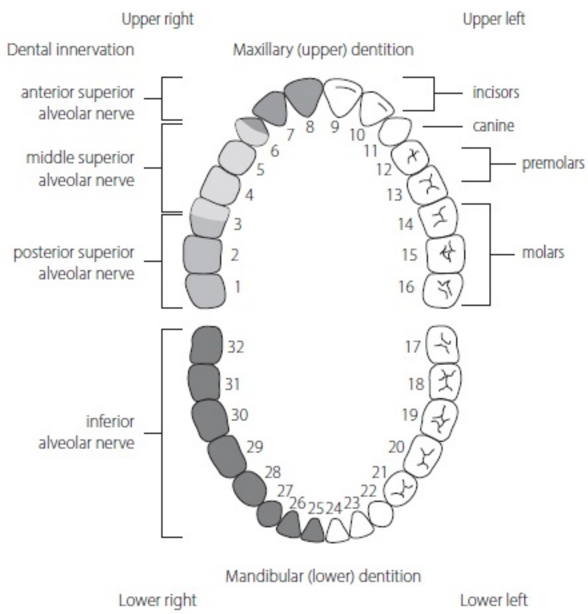


Figure 309.2 Dental block

Inferior alveolar nerve block

Place a metal retractor or wooden tongue blade into coronoid notch (bony curvature on anterior border of the mandibular ramus). Approach patient from contralateral side with syringe angled between the 1^{st} and 2^{nd} premolars.

- Insert needle into soft tissue until it contacts periosteum of the medial aspect of the ramus. **Do not inject anaesthetic except when this contact is confirmed.** Carotid artery and jugular vein course immediately posterior
- Withdraw the needle 1 mm and aspirate to confirm extravascular placement
- Inject 2–4 ml of anaesthetic solution and massage for 10 seconds

Posterior superior alveolar nerve block

Insertion site: a palpable buccal soft spot posterior to the zygomatic arch and superior to the 2^{nd} molar.

- Insert needle into mucobuccal intersection at 45o angle to a depth of 2.5 cm. Needle tip should be directed toward inner corner of contralateral eye
- **Aspiration is important for this nerve block as this area of the maxilla is highly vascular**
- Inject 2–2.5 ml of local anaesthetic

Middle superior alveolar nerve block

This nerve should be anaesthetised along with the posterior superior alveolar nerve to ensure adequate coverage of the 1^{st} molar.

- Insert needle at 45° at mucobuccal fold between 2^{nd} premolar and 1^{st} molar
- Advance 1–1.5 cm, aspirate
- Inject 2–3 ml of local anaesthetic

Anterior superior alveolar nerve block

- Insert needle at 45° at the mucobuccal fold above the apex of the canine tooth
- Advance 1–1.5 cm, aspirate
- Inject 2–3 ml of local anaesthetic

Supraperiosteal nerve block

- Insert needle at 45o at mucobuccal fold at site of affected tooth, advance 1–1.5 cm and aspirate

• Inject 2–3 ml of local anaesthetic

Aftercare

Potential complications

See consent above.

Documentation

Neurovascular status before and after procedure, region anesthetised, medications, outcome.

Disposition

For underlying condition. In dental cases, refer to a dentist as needed.

310 Basics of mechanical ventilation

Terminology and techniques

Mechanical ventilators generate flow. Clinicians may set how each breath is initiated (by patient or machine), how many breaths are delivered, how quickly each breath is delivered, what volume and pressure limits are acceptable, when each breath should be terminated, and whether to assist or not when the patient attempts a spontaneous breath.

Ventilation may be:

- Non-invasive: face or nose mask with nothing in the airway, e.g. CPAP, BiPAP, NIPPV
- Invasive: definitive, secured airway (such as endotracheal tube)

The mode of ventilation refers to how the machine initiates, delivers and times each breath cycle. Some common examples include:

- CMV: controlled, mandatory ventilation:
  - The machine delivers the set amount of breaths per minute, using the set parameters for flow and pressure, without regard for spontaneous patient breaths
  - Consider in patients with no spontaneous breathing (paralysed).
- ACV: assist control ventilation:
  - The machine achieves the set respiratory rate by assisting each patient breathing attempt with the specified volume or pressure and delivering CMV breaths to make up the difference
  - e.g. set rate = 15, with patient spontaneous respiratory rate = 5: machine supports 5 patient breaths and delivers additional 10 breaths per minute
  - ACV avoids air hunger, but may substantially over-ventilate and drop CO_2 in patients who have central tachypnoea (tachypnoea of head injury, for example), as every breath will be supported
- SIMV: synchronised intermittent mandatory ventilation:
  - Similar to ACV, but the machine will not assist patient's spontaneous breaths
  - Synchronisation means the machine will not deliver a mechanical breath during a patient breath, thereby reducing the risk for stacking and hyperinflation
  - Set rate = 15, with patient spontaneous respiratory rate = 5: patient will get their own intrinsic tidal volume for 5 breaths, and 10 additional machine breaths. Some SIMV settings will assist patient breaths if they begin near the time a machine breath is due
  - Avoids over-ventilation and breathstacking, because patient's own breaths are not assisted, but can under-ventilate (causing hypercapnoea and fatigue) in patients breathing at normal rates with low tidal volumes. SIMV does not guarantee tidal volume. Increases work of breathing for the patient

The control of ventilation refers to how the machine delivers and terminates each breath. Some common examples:

- Volume controlled:
  - The machine will deliver a set volume, regardless of airway pressure. This strategy risks barotrauma
- Pressure controlled:
  - The machine will deliver flow until a set pressure is met, regardless of volume. This strategy risks volutrauma

- Dual control:
  - » The machine will deliver a set volume, but terminate the breath prematurely if a pre-set pressure limit is reached (or vice versa)
  - » This is the preferred, safer, control strategy

Modern ventilators often have the ability to combine different modes and controls, making it possible to better tailor the ventilation strategy to each patient's needs.

Basic set up

- In principal, set or adjust the following parameters:
- Inspired O_2 concentration (FiO_2):
  - » Start at 100%. Consider titrating the FiO_2 downwards as soon as the patient's condition and oxygenation improves. Do not set concentrations below 40% for ill patients
- Respiratory rate:
  - » Initial rate should be set at 10–14 bpm (or at a rate appropriate for age and underlying disease process and condition of lungs) and adjusted to achieve oxygenation and CO_2/pH goals
  - » If the patient is already breathing at a higher rate that is physiologically necessary and not compensated by oxygenation (e.g. in severe acidosis) consider setting initial rate closer to patient rate while patient is paralysed or heavily sedated and unable to drive rate. Rational use of ABG is an important part of monitoring and adjusting ventilation. A ventilator will not be able to 'keep up' with ventilation needs above 35 bpm
  - » Some patients may require initial higher rates (restrictive lung disease, severe hypercarbia, acidaemia) but do not exceed 35 bpm
  - » Some may require lower rates (patients with obstructive airways disease, i.e. asthma and COPD)
- Tidal volume:
  - » Initial tidal volume should be set to 8 ml/kg (range 6–10, higher in patients with healthy lungs, lower in patients with concern for acute lung injury) and adjusted to reach O_2 and CO_2/pH goals
- Pressure limit:
  - » Set initial peak pressure limit to 30 cm H_2O. Short periods of higher pressures may be allowed, up to 40 cm H_2O, but should prompt rapid identification and reversal of the cause of the high pressures. Aim for plateau pressures well under 30 cm H_2O
- Inspiratory: expiratory time ratio (I:E time):
  - » Expiratory time should almost always exceed inspiratory time. Set the I:E ratio to 1:2 initially and adjust as needed. Adequate expiratory time is essential in asthma and COPD to avoid breathstacking and increased pressures that can impede venous return. I:E ratios of 1:2–1:6 or even greater may be required
- PEEP:
  - » Minimum PEEP should be 5 cm H_2O. Some patients may initially require higher PEEP (i.e. patients with pulmonary oedema) but be careful when exceeding 10 cm H_2O as this may impede venous return and result in hypotension or even shock

There are many machines, modes and techniques to achieve these settings. Some machines may require you to set inspiratory pressure and then deliver whatever volume is needed to achieve this or vice versa. Most modern machines allow you to manually adjust all these settings while calculating safe margins and optimal values. Get to know your machine.

311 Wound management and suturing

Indications and contraindications for suturing

Indications

To close clean, gaping wounds < 6 hrs old (< 24 hrs for facial wounds)

Contraindications

Do not suture infected or puncture wounds (absolute); wounds > 6 hrs prior to presentation, bites (relative).

Preparation

- History: immunocompromised state, smoking, medications that alter wound healing (e.g. steroids), tetanus immunisation
- Physical examination of the area, including detailed distal neurovascular status

Consent

Risks include scar (risk can be decreased by keeping area covered, out of the sun), infection, dehiscence, missed foreign body or fracture, and missed tendon or nerve injury. Benefits include aesthetic improvement, and avoidance of infection. Alternatives include dressings, and alternative wound closure techniques.

Equipment

- Clean gloves (do not need to be sterile), eye protection
- Irrigation supplies (large syringe or small feeding tube)
- Clean water for irrigation (does not need to be sterile)
- Suture kit (haemostat, forceps, scissors, needle holder)
- Wound closure agent (sutures, steri-strips, skin adhesive)
- Dressing supplies

Types of suture material

- Non-absorbable: nylon, polypropylene, silk
- Absorbable: gut, polyglactin (Vicryl)

Table 311.1 Suture size and removal time by location

Location of wound	Size of suture	Number of days until removal
Face	5.0–6.0	5–7
Hand	4.0	10–14
Elsewhere	4.0	7–10

Procedure

1. Anaesthetise the wound; consider blocks to minimise swelling and tissue distortion at the site of the wound.
2. Irrigation – most effective way to minimise infection risk.
3. Explore: look for foreign bodies and extension (control bleeding to improve inspection) into joint capsule, evaluate for fractures and tendon injury. If unable to visualise the base of the wound or otherwise concerned, use US or XR to evaluate for foreign body.
4. Debride any devascularised tissue.
5. If indicated, close the wound with sutures or an alternative method.

Suturing

- Primary closure: the best option. If needed, place deep layer of suture (non-absorbable). Place simple interrupted sutures through the skin (most common) to approximate wound edges
- Secondary closure: if wound contraindications for primary closure, you may leave the wound open. Excellent wound care is essential. Remember that the scar will be more significant
- Delayed primary closure: for wounds that have a high risk for infection – dirty wounds, bite wounds. After irrigating, apply wet-to-dry dressing. Ask the patient to keep the wound moist and clean and to return in 48–72 hours. If there is no sign of infection at that point, close it with sutures

Alternatives to suturing

- Skin stapler
- Tissue adhesive (e.g. Dermabond)
- Adhesive tapes (Steri-strips)
- Hair tying for scalp wounds

Aftercare

- Update tetanus if needed. If wound is under tension and over an extensor surface, consider removable splint to improve wound healing
- For bite wounds, dirty wounds, wounds in immunocompromised hosts, wounds with exposed tendons: consider antibiotic prophylaxis
- For bite wounds from unvaccinated animals: consider rabies prophylaxis
- Keep clean and dry, apply antibiotic ointment (not for wounds closed with tissue adhesive because it will dissolve with petroleum based products)
- Explain return precautions (signs of infection, dehiscence)

Potential complications

See consent (above).

Documentation

Detailed neurovascular exam, procedure, complications.

Disposition

Admit as per the underlying condition.

312 Vascular access

Accurate knowledge of the underlying anatomy, concurrent use of clinical aids such as US, and practical dexterity gained through experience increases the likelihood of success and minimises complications.

Obtaining vascular access in a sick child can be challenging for any provider. Developing a stepwise protocol for vascular access can alleviate stress, reduce time to access, and improve patient outcomes. Where available and appropriate, application of topical local anaesthetic cream can facilitate access.

An IO line is an infusion needle placed into the bone marrow cavity, which is continuous with venous circulation and can be used to infuse fluids and drugs for a short time. Aim to establish formal venous access as soon as possible.

Indications and contraindications

Indications

Central venous access

- **Monitoring of central venous pressure (CVP)**
- **Volume resuscitation**: higher flow rates can be achieved with large bore peripheral IV (14G). For large volumes, use large bore central access (8Fr)
- **Emergency venous access**: anatomic landmarks and predictable location of femoral and subclavian veins make them great targets in emergencies
- **Difficulty obtaining peripheral access**: IV drug abusers, morbidly obese patients, renal dialysis patients, patients with significant TBSA burns
- **To assist with another procedure**: pulmonary artery catheter or transvenous pacemaker; cardiac catheterisation; placement of a dialysis catheter

IO line

• Life-threatening situations, when IV access fails (2 attempts or <90 seconds)
• In cardiac arrest, as first-line strategy, particularly in children and when vascular access is difficult

Contraindications

• Central line: distortion of local anatomy, vessel vasculitis, combative patient, previous long-term cannulation of the vessel, suspected proximal vascular injury, anticoagulation therapy, infection overlying insertion site
• IO line: bone disease (e.g. osteogenesis imperfecta), fracture in chosen bone, previous IO attempt in chosen bone, infection or burn over insertion site

Preparation

Consent

Risks include infection, bleeding, PTX, air embolism, nerve injury.
Benefits include definitive intravenous access. **Alternatives** include peripheral access and alternate treatment plans (for specific central line indications).
IO line is an emergent procedure: consent is implied.

Equipment

Central venous access

• Sterile protective equipment, central venous access kit, US (if available)

IO line

• Alcohol swabs
• Syringes (5 ml to aspirate, 20 ml to administer boluses)
• Short IV tubing extension set, 3-way stop cock
• **Mechanical IO device**: use provided instructions
• **Manual IO placement**: if no device is available use one of the following needles (in decreasing order of preference):
  » 18G bone-marrow aspiration needle
  » 18G SHORT spinal needle
  » 21G plain needle (or 18G needle for child > 2–3 years)

Procedure

Central venous access

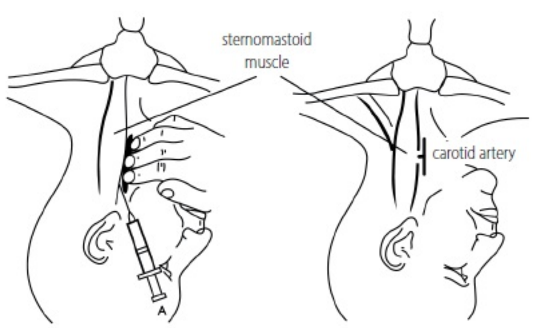


Figure 312.1 Internal jugular cannulation

Source: Brown & Wyatt. 2008. *Oxford American Handbook of Emergency Medicine*. By permission of Oxford University Press, USA.

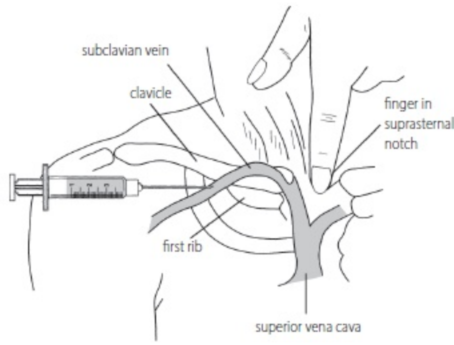


Figure 312.2 Subclavian vein cannulation

Source: Brown & Wyatt. 2008. Oxford American Handbook of Emergency Medicine. By permission of Oxford University Press, USA.

- Widely prep the skin over the desired site with a cleansing solution:
  - » If attempting a line in the upper chest, prep all three sites: internal jugular, and infraclavicular and supraclavicular subclavian approaches
- Apply sterile drapes
- Use landmarks or US to identify the intended vessel
- Insert a small needle into the vessel. Once blood is easily drawn into the syringe, stabilise the needle with your other hand
- Remove the syringe from the needle and insert guidewire into the needle
- **The guidewire should always be visible and held by the practitioner** When placing a line in the upper chest or neck, the wire should be advanced while observing the monitor. Watch for PVCs or ventricular dysrhythmias: pull the wire back until ECG normalises
- Once the wire has been advanced to the desired position, remove the insertion needle. **Keep a firm hold of guidewire at all times.** Once needle removed, make a small incision at the guidewire site using #11 blade
- Advance introducer over the guidewire. Once you reach the skin, grasp the introducer close to its tip and advance into subcutaneous tissue to create the appropriate track. Twist the introducer in the same plane as the vessel. Once the track is created, remove the introducer
- **While holding guidewire**, start to advance the catheter over the guidewire. When there is approximately 3 cm of the wire exposed at the insertion site, withdraw the wire from the vessel towards the catheter
- Once the tip of the guidewire is removed from the end of the catheter, grab hold of the wire at this end. Gently advance the catheter into the vessel over the remaining guidewire
- Remove the guidewire completely
- Draw back on all available ports of your catheter to ensure good flow, and flush them with sterile saline
- Suture the line in place
- Obtain post-procedure CXR (evaluate line placement, exclude PTX)

IO line

- Choose infusion site: 1st choice is the upper anteromedial tibia
  - » **Upper anterior-medial tibia**: 2 cm below tibial tuberosity (shorter distance in smaller babies) and 1–2 cm medially; flat part of the bone
  - » **Lower femur**: Anterolateral surface 3 cm above the lateral condyle
  - » **Upper humerus**: Laterally into the greater tuberosity
- Clean skin over chosen site. *This is often a life-saving procedure therefore local anaesthetic may not be required*
- Insert needle 90o to bone surface with a firm ‘screwing and drilling’ motion
- Continued to advance until a sudden decrease in resistance (‘a give’) is felt as the cortex is penetrated. **Feeling may be subtle in very small infants**
- Remove stylet from needle if there is one. Blood may come up into the hub – this is a good sign of correct placement

- If you do not see this ‘flashback’ you may still be in correct place. Attach syringe and attempt to aspirate; sometimes nothing is aspirated, but if:
  » You felt a clear ‘give’ as you entered the cortex, and
  » The needle is firmly standing upright (not swaying when touched)

  then it is worth trying to infuse through the line, watching carefully for infiltration
- To prevent repeated direct handling of IO needle: attach flushed IV extension set (ideally with 3-way stop cock) to needle-hub
- Drugs and fluid boluses will need to be pushed in with a syringe using a 3-way stopcock and push-pull method; pressure in the cavity does not allow rapid free-flow
- Keep checking surrounding tissues for evidence of infiltration or displacement.
- Secure IOL:
  » Methods include use of: plastic umbilical cord clamp or trouser-leg taping (piece 1-inch tape split to halfway point into two ‘trousers-legs’. Trouser-top (unsplit) part applied to child’s leg, then one trouser leg wrapped around and up IO needle with remaining trouser-leg directly onto child’s leg. Repeat with another piece of tape cut and applied in same way from other side of needle
  » If child transported with IOL *in situ*, apply obvious dressing (e.g. surround with cotton-balls or folded-gauze) so it is not covered and neglected
  » **Never strap circumferentially**; distal circulation may be compromised

Paediatric vascular access

Peripheral venous access

Sites: dorsal hand, forearm, median cubital vein, greater saphenous vein, dorsal foot, scalp, and external jugular vein.

Procedure:

- Apply a tourniquet proximal to the site of the vein
  » Scalp vein: place a small rubber band around the head just above the ears
  » External jugular: place patient head down
- Clean the skin over the vein
- Pull the skin taut with non-dominant hand
- Puncture the skin with the needle at a 30-degree angle, keeping the bevel up
- Once blood flow is seen through the tubing, advance the tip a few mm to ensure placement in the vein; thread the catheter over the needle into the vein

Complications: haematoma, infection, thrombosis, phlebitis, and infiltration of medicine into the tissues.

Peripheral venous cutdown

Site: greater saphenous vein.

Procedure:

- Immobilise the leg with foot rotated laterally
- Prepare skin over medial ankle in sterile fashion. Anaesthetise with 1% lidocaine
- Make a transverse incision 1–2 cm above and anterior to the medial malleolus.
- Dissect through the subcutaneous tissue with a curved haemostat, parallel to the course of the vein. Carefully separate the vein from surrounding tissues
- Tie a small absorbable suture on the distal end of the vein and pull vein up by applying traction
- Place suture around the proximal end of the vein and clamp the end of the suture with a haemostat
- Pull the vein taut by applying traction on the clamp with the proximal suture
- Advance a catheter into the vessel
- Tie the proximal end of the suture around the part of the vein containing the catheter
- Suture the catheter to the skin and close the remaining incision
- Place a sterile dressing over the IV site

Complications: haematoma, bleeding, transection of the vein, infection, thrombophlebitis, injury to adjacent structures (tendons and sensory nerves).

Umbilical venous access

- Create a sterile field around the umbilicus
- Tie umbilical tape or sterile string around the base of the cord
- Use a scalpel to cut the cord 2–4 cm from the abdomen, parallel to the umbilical stump
- Identify the umbilical vein (cord contains three vessels: one larger thin-walled vein and two small thick-walled arteries)
- Grab the edge of the umbilicus with haemostats to stabilise and apply traction while threading a 3.5 or 5.0 French catheter through the umbilical vein
- Advance the catheter below the skin and aspirate to confirm placement. Gently flush with sterile saline to confirm patency. Secure in place with suture or tape

Complications: bleeding, infection, air embolism, creation of false tract or vessel perforation, thrombosis, liver abscess or necrosis.

Table 312.1 Venous catheter sizes by age

Age	Needle gauge	French size	Length
Newborn	26, 24	3.0–4.0	5–12 cm
Infant (<1 year)	24	4.0	5–12 cm
1–8 years	22, 20, 18	4.0–5.0	5–25 cm
> 8 years	20, 18	5.0 – 8.0	5–30 cm

Other options

Consider nasogastric tube if unable to quickly obtain access.

Aftercare

Potential complications

See consent (above). Iatrogenic fracture seen with IO lines.

Documentation

Document site, technique, number of attempts, complications.

Disposition

Admit. Aim to replace IO with a definitive IV line within 24 hours.

313 Urethral catheterisation

Facilitates direct drainage of the urinary bladder; used for diagnostic and therapeutic purposes. May be inserted as an in-and-out procedure for immediate drainage, or left indwelling.

Indications and contraindications

Diagnostic or therapeutic

- Collection of sterile urine specimen
- Monitoring of urine output
- Imaging of the urinary tract as part of urologic studies
- Acute urinary retention (e.g. benign prostatic hypertrophy, blood clots)
- Chronic obstruction with hydronephrosis
- Continuous bladder irrigation
- Urinary incontinence

Contraindications

- Absolute: traumatic injury to the lower urinary tract (e.g. urethral tear – suspected in male patients with a pelvic or straddle-type injury; high-riding or boggy prostate, perineal haematoma, or blood at the meatus). When suspected, do a retrograde urethrogram to rule out a urethral tear prior to placing a catheter
- Relative: acute prostatitis or urethritis or known tight urethral strictures

Preparation

Consent

Risks include nosocomial UTI (especially when catheter is *in situ* for prolonged periods), urethral or bladder injury, haematuria, retained catheter, and inability to place catheter. Benefits include symptom relief, and facilitation of diagnosis. Alternatives include aspiration, suprapubic catheterisation, and surgery.

Equipment

- Sterile Foley catheter
- Betadine solution (povidone iodine) or other antiseptic solution
- Lidocaine gel (2%)
- Sterile cotton balls or gauze, drapes and gloves
- Water-soluble lubrication gel
- Prefilled 10 ml saline syringe
- Sterile urine collection bag

Procedure

The procedure varies minimally between male and females; the basic principles of aseptic precautions and positioning are the same. In uncircumcised males, complete control of the foreskin is paramount.

1. Preliminary hand wash and wearing of cap and mask recommended.
2. Place patient supine with legs slightly apart (male) and with legs apart and knees flexed (female).
3. Cleanse external genitalia with an antiseptic solution.
4. From this point all procedures are done with sterile gloves.
5. Cleanse genitalia using Betadine solution and place sterile drapes exposing external genitalia.
6. Check Foley catheter balloon by introducing 5 ml of water into the balloon and then deflating.
7. Lubricate the sterile catheter with water-soluble lubricant.

Male

- Hold penis with your non-dominant hand upright, away from scrotum
- Hold catheter firmly with dominant hand, gently pass well lubricated catheter through external urethral meatus. Gently and gradually advance catheter until it passes into bladder: should see urine flow through catheter
- Advance length of catheter and then inflate bulb with 5–10 ml saline. Gently pull back until inflated balloon purchases
- Connect Foley catheter to collecting bag. Secure the tube/bag to the medial aspect of one of the thighs using tape

Female

- Separate the labia with the thumb and index fingers of the non-dominant hand and identify the urethral meatus
- Gently advance previously lubricated catheter through the meatus into the bladder. Remember the female urethra is short (~4.0 cm)
- Once urine is seen coming out of the tube, advance, inflate and secure as described above

Aftercare

Keep catheter, urine bag and genitalia clean; change catheters when necessary.

Potential complications

See consent above.

Documentation

Indication, size of catheter, amount of water to inflate the balloon, quantity and composition of urine drained, any difficulty encountered during the procedure.

Disposition

Admit as per the underlying condition.

314 Suprapubic catheterisation

Facilitates trans-cutaneous drainage of the urinary bladder when urethral catheterisation is not possible

Indications and contraindications

Indications

Urethral injury or obstruction

Contraindications

Relative: ability to pass urethral catheter, absence of an easily palpable or US-localised distended urinary bladder, coagulopathy, prior lower abdominal or pelvic surgery, pelvic cancer, pelvic radiation, overlying infection.

Preparation

Consent

Risks include frank haematuria (typically transient), insertion site cellulitis, ureteral injury, obstruction, bowel perforation, visceral injuries, and inability to pass catheter. Benefits include symptomatic relief. Alternatives include surgical cystostomy.

Equipment

- Sterile gloves, gauze and drapes
- Antiseptic solution
- Local anaesthetic (2%)
- 10 ml and 60 ml syringes
- 18G and 25G needles
- Scalpel
- Percutaneous suprapubic catheter set (paediatric: 8–10F; adult: 12–16F). *Can use a central venous catheter kit and Seldinger technique too*)
  - » Needle obturator
  - » Malecot catheter
  - » Connecting tube
  - » One-way stopcock
- Sterile urine collection bag
- Drain sponges
- Skin tape or nylon suture (3–0) with a needle driver

Procedure

1. Provide adequate parenteral analgesia.
2. Palpate the distended bladder and mark the insertion site at the midline, two fingers (4–5 cm) above the pubic symphysis.
   - Use US ◇, where available, to verify bladder location and ensure no loops of bowel between the abdominal wall and the bladder.
3. Apply antiseptic solution and drapes from the pubis to the umbilicus.
4. Anaesthetise the insertion site using the 10 ml syringe and the 25G needle.
5. Advance the needle through the skin, rectus sheath, and retropubic space, while alternating injection and aspiration, until urine enters the syringe. Note the direction and depth required to enter the bladder.
6. Using the scalpel, make a 4 mm stab incision at the insertion site.
7. Insert the needle obturator into the Malecot catheter and lock it into the port by twisting it so that the needle tip projects 2.5 mm from the distal end of the catheter.
8. Connect the 60 ml syringe to the port of the needle obturator.
9. Place the tip of the catheter-obturator unit into the skin incision and direct it caudally at a 20–30° angle.
   - Place your non-dominant hand on the lower abdominal wall to stabilise the unit between the thumb and index fingers
   - While aspirating, use your dominant hand to advance the unit until urine enters the syringe
   - Once this occurs, advance the unit an additional 3–4 cm into the bladder
10. While securing the unit with the non-dominant hand, unscrew the obturator from the catheter, unlocking the needle obturator from the catheter.
11. Advance the catheter approx 5 cm over the obturator and then completely withdraw the obturator needle, advancing the catheter over the needle.
12. Connect the extension tubing to the catheter and connect the tubing to a urine collection bag.
13. Gently withdraw the catheter to lodge the wings against the bladder wall.
13. Undrape the patient and apply a dressing around the catheter insertion site.
15. Tape or suture the catheter to the skin.

Aftercare

Do not change a newly inserted catheter for four weeks (unless infection or other complication); this allows the catheter tract to become established. Subsequently inserted tubes should be changed at least once a month to decrease infection rate.

Potential complications

See consent above.

Documentation

Document indication, catheter size, amount of water to inflate balloon, quantity and composition of urine drained, complications.

Disposition

Refer to urologist.

315 Lumbar puncture

Perform neurologic imaging prior to LP for patients with signs of increased ICP.

Indications and contraindications

Indications

- Diagnose subarachnoid haemorrhage

• Diagnose meningitis (never delay antibiotics for LP)

Contraindications

• Tissue infection at the puncture site
• Increased ICP (space-occupying lesion or obstructive hydrocephalus)
• Spinal epidural abscess
• Brain abscess
• Bleeding diathesis
• Meningococcal sepsis
• Severe systemic illness that precludes positioning, particularly in babies

Preparation

Consent

Risks include infection (meningitis), post-LP headache, spinal haematoma, and cerebral hernation. Benefits include definitive diagnosis. Alternatives include empiric management.

Equipment

• Antiseptic solution
• Sterile draping, gauze and gloves
• Spinal needle (20 or 22G, 3.5 inch (88 mm) for adults, 2.5 inch (63 mm) for children, 1.5 inch (35 mm) for infants)
• Collection tubes
• Manometer
• 3-way stopcock
• Lidocaine
• Syringe with 22G–25G needles
• Dressing

Procedure

1. Proper positioning is essential: lateral decubitus position with knees drawn towards the chest, effectively arching the lower back towards the provider. A hunched-over seated position is an alternative (but CSF pressure reading in this position is not accurate).
2. Locate the midline of the back at the level of the posterior superior iliac crests, and cleanse the area with an antiseptic solution.
3. Tuck a sterile towel or commercial drape kit between the patient and the bed.
4. Palpate the L3-L4 or L4-L5 interspace and infiltrate the skin and subcutaneous tissue generously with lidocaine.
5. Use a spinal needle to enter the L3-L4 or L4-L5 interspace at midline. The needle bevel should be towards the patient’s flank. The needle should parallel to the bed, with a slight cephalad angle.
6. Advance the needle slowly. As it enters the subarachnoid space the provider often feels a ‘pop’. Retract the stylet frequently to check for CSF.
7. If bone is encountered, withdraw the needle to the level of the subcutaneous tissue, palpate landmarks and redirect the needle. Often, a more cephalad angle is helpful.
8. When CSF is encountered attach the manometer and 3-way stopcock. Record the opening pressure.
9. Collect the fluid from the manometer in tube number one. Remove the manometer and continue fluid collection in tubes two through four. Collect sufficient volume for necessary testing (typically at least 1–2 ml per tube).
10. Replace the stylet into the needle prior to withdrawing it.
11. Place a dressing over the site.

Aftercare

Potential complications

See consent (above). Post-LP headache: if symptoms are severe or prolonged consider caffeine and/or a blood patch.

Interpretation of results

Table 315.1 Classic CSF findings

	RBCs (per mm^3)	WBC (per mm^3)	Glucose (mg/dl)	Protein (mg/dl)	Opening pressure (cm H_2O)	Appear-ance
Normal	<10	<5	2/3 of serum	20–45	5–20	Clear
Bacterial	Normal	↑ (PMN)	↓	↑	↑	Cloudy
Viral	Normal or ↑	↑ (L)	Normal	Normal or ↑	Normal or ↑	Clear
Fungal	Normal	↑ (L)	↓	↑	↑	Clear
SAH	↑	↑	Normal	↑	Normal or ↑	Yellow or Red

L = Lymphocytes, PMN = Polymorphonuclear neutrophils, Increased = ↑, Decreased = ↓

316 Abscess incision and drainage

An abscess is a collection of pus that often presents with tenderness, redness and warmth of overlying skin. Antibiotics do not penetrate well and incision and drainage is usually necessary and sufficient treatment. Blood work and wound cultures are not routinely indicated, though may be useful in high risk patients. Differential includes: sebaceous cyst, Bartholin's gland abscess (at vaginal wall), hidradenitis suppurativa (axilla and groin), and atypical cutaneous infections (e.g. *Sporotrichosis*, *Leishmanisasis*, *Tularemia*, *Myiasis*, *Botryomycosis*, Non-TB *Mycobacteria*). US can be useful to identify and locate abscesses, but cannot rule out abscess, as denser, organised purulent material may appear at same density as surrounding tissues.

Indications and contraindications

Indications

• Drainage of a localised collection of pus

Contraindications

• Proximity to large vascular structures, neurovascular bundle or if vascular injury suspected
• Located at corners of mouth or bridge of nose
• Large or complicated abscesses may require OT with GA

Preparation

Determine location, size, extent, proximity to neurovascular structures, and foreign body presence. US or XR may be helpful ◇. Ask about time course of skin findings, systemic symptoms (fever, chills), history of previous abscesses, immune status and possibility of occult foreign body (sanding or grinding work), and history of trauma, including human/animal bite.

Consent

Risks include spread of infection, recurrence, bleeding, damage of nearby organs. Benefits include relief of symptoms. Alternatives include antibiotic therapy.

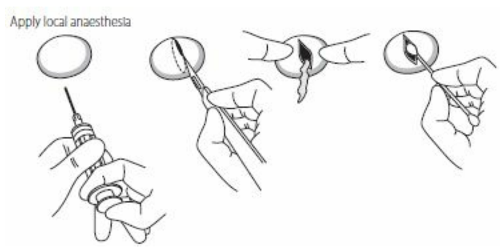
Equipment

Skin disinfectant, local or regional anaesthesia, scalpel, haemostat or cotton swab to sweep cavity, and gauze or iodoform packing. Suction may be helpful for larger abscess.

Procedure (Figure 316.1)

1. Clean wound with antiseptic solution.
2. Apply local or regional anaesthesia.
3. Incise at the most fluctuant area; extend incision ⅔ length of abscess.
4. Express pus from cavity.
5. Sweep cavity with haemostat or cotton swab to break any loculations.
6. Irrigate abscess cavity.
7. Apply sterile bandage.

Figure 316.1 Incision and drainage



Special considerations

- Sebaceous cyst abscess: must remove cyst wall to prevent recurrence
- Bartholin's gland abscess: place catheter drain instead of gauze packing. May consider marsupialisation procedure to reduce rate of recurrence.

Aftercare

Potential complications

See consent (above).

Documentation

- Size and appearance of initial abscess
- Procedure: incision length, number of gauze used in packing, time and date.

Disposition

- Uncomplicated abscesses: discharge with follow-up in 24–48 h to change packing
- Surgical drainage with packing changes is usually adequate treatment for simple abscess, but indications for antibiotics include recurrent abscess, significant surrounding cellulitis, signs of systemic inflammation, immuncompromise, or abscess of hand or foot
- Complicating factors that may require admission or OT intervention include: large abscesses, rapid spread, excessive bleeding, or complicated site (i.e. perirectal abscess or near neurovasculature)

317 Drainage of a peritonsillar abscess

Indications and contraindications

Indications

- Peritonsillar abscess (PTA)
- Use I and D where needle aspiration fails

Contraindications

- Absolute: malignancy, vascular malformation
- Relative: severe trismus, uncooperative patient

Preparation

- Place patient upright and seated with posterior head support
- Sedation and analgesia may help, but over-sedation prevents patient compliance
- Consider and exclude airway compromise and deep space infections of neck

Consent

Risks include failure, aspiration of abscess contents into airway, and internal carotid artery injury (located 2.5 cm anterolateral to PTA). Benefits include symptomatic relief, and accurate diagnosis. Alternatives include antibiotic therapy.

Equipment

- Gloves, headlamp or other light source, tongue depressor, sterile gauze, 27G needle, 5 ml syringe, saline, local anaesthetic with adrenaline, topical anaesthetic spray, oral suction, parenteral anxiolytics or narcotics (if available)
- US with linear or phased array endocavitary probe and cover ◇
- For needle aspiration: 18–20G needle with needle guard, 10–20 ml syringe. Cut and replace needle guard at distal end to leave only distal 1 cm of needle exposed (helps prevent carotid artery puncture)
- For I and D: medical tape, scalpel, Kelly clamp, suction. Tape the scalpel so that only most distal 1 cm of blade is exposed (rationale as above)

Procedure

1. Retract cheek. Use tongue depressor or laryngoscope to improve visualisation.
2. Anaesthetise area to be aspirated/incised with local infiltration of 1–2 ml using 27G needle on 5 ml syringe. Allow 5–10 minutes for full effect. Consider pre-treatment with topical anaesthetic spray.
   - For aspiration: if using US, place probe in mouth over abscess (abscess will appear as cystic or heterogeneous mass with irregular border); insert needle with US guidance and aspirate with advancement. If US not available, insert needle at most fluctuant or prominent part of abscess, aspirating as needle is advanced in sagittal plane. DO NOT advance laterally toward carotid sheath. If no aspiration at 1 cm depth, remove and repeat 1 cm superior or inferior. If no pus is aspirated after three attempts, consider I and D or alternative diagnosis. If pus is aspirated, remove as much as possible (usually 2–6 ml)
   - For I and D: make 0.5 cm incision in anterior-posterior direction at area of greatest fluctuance or at prior aspiration site if performed. Perform suction to remove pus. Insert Kelly clamp to gently break up loculations. Repeat suctioning. Packing not needed
3. Give patient saline solution, and ask to rinse and gargle. Repeat 3–4 times

Aftercare

Observe for at least one hour. Antibiotics 5–7 days – penicillin, clindamycin, cephalosporins, amoxicillin/clavulanate.

Steroids can reduce severity of pain/inflammation post-drainage.

Potential complications

See consent above.

Interpretation of results

If aspiration fails after three attempts, or if I and D reveals no pus, consider alternative diagnosis.

Documentation

Document use of US, number of attempts, amount and description of fluid obtained, blood loss (if significant), complications.

Disposition

Discharge if systematically well, no airway compromise, can swallow. Otherwise, admit.

318 Nasal packing

Anterior nosebleeds are more common than posterior. 90% of anterior bleeds occur within Kiesselbach’s plexus; posterior nosebleeds can result in significant blood loss and are more difficult to control.

Indications and contraindications

Indications

- Continuing or recurrent epistaxis not controlled with other techniques (such as cautery)
- Posterior packing indicated when epistaxis not controlled by anterior packing

Contraindications

- Absolute: disrupted anatomy, basilar skull fracture (intranasal balloon may migrate into skull cavity)
- Relative: nasal haematoma.

Preparation

Consent

Risks include local tissue damage, infection, airway compromise and arrhythmia (posterior packing). Benefits include symptom control and definitive diagnosis. Alternatives include direct pressure, cautery and embolisation.

Equipment

- Nasal speculum
- Bayonet forceps
- Cotton swabs
- Topical anaesthetic
- Antibiotic ointment that provides Gram positive coverage (i.e. bacitracin)
- Packing material (ribbon gauze, nasal tampons or nasal balloon catheters)
- Tape
- Topical vasoconstrictive agent (e.g. oxymetazoline) ◇

Procedure

Topical anaesthesia and vasoconstriction

- Administer a local vasoconstrictor/decongestant spray
- Anaesthetise nose before any attempts at packing
- Soak two cotton swabs in topical anaesthetic and place in affected naris. Alternative: use local anaesthetic spray. Remove before packing

Gauze packing

- Most difficult method
- Grasp gauze with forceps 10 cm from end and advance into nasal cavity as far as possible through nasal speculum. Remove speculum and forceps. Replace speculum over layer of gauze and pack gauze to nasal floor. Repeat with another 10 cm of gauze on top of previous layer, continuing in accordion fashion until tightly packed.

Nasal tampon

- Coat nasal tampon with antibiotic ointment. Insert entire tampon along floor of nasal cavity. Secure string to cheek with tape. Apply topical vascoconstrictive agent onto the tampon

Nasal balloon catheters

- Begin with 5 cm (anterior) balloon catheter; replace with 7.5 cm (posterior) catheter if haemostasis not achieved
- Lubricate balloon catheter by soaking in sterile water for 30 seconds. Insert catheter along floor of nasal cavity and inflate catheter cuff. Secure string to cheek with tape

Considerations for posterior epistaxis

- If posterior nasal balloon catheter has two balloons, advance catheter, then inflate posterior balloon. Retract catheter gently until balloon lodges against posterior choana. Inflate anterior balloon and secure to cheek
- A 14 Fr Foley catheter can be used. The tip should be trimmed carefully to avoid irritation. Insert Foley along floor of nose until visible in oropharynx. Partially fill the balloon and retract until balloon lodges against posterior choana. Then completely fill the balloon
- A red rubber catheter can be used. Tie two silk ties around middle of gauze roll and extend them in opposite directions. Pass catheter through nose into oropharynx and grasp with forceps, pulling end out of mouth. Attach one tie to catheter and pull gauze from mouth through nose. Tape tie from oropharynx to cheek for removal. Use tie from nostril to maintain position. Pack anterior nose with gauze

Aftercare

Potential complications

See consent above.

Antibiotics

Not routinely advised; prophylactic antibiotics for *Staph/Strep* with posterior packing (penicillin 500 mg Q6h OR amoxicillin-clavulanate 500 mg TID or cephalexin 500 mg QID).

Disposition

- Anterior packing: discharge and review (ideally with ENT) within 24–48 hours
- Posterior packing: admit

319 Lateral canthotomy

An emergency procedure to decompress orbital compartment syndrome (OCS), restore retinal artery blood flow, and save vision. OCS can develop from retrobulbar haemorrhage after orbital trauma.

Indications and contraindications

Indications

- Primary: decreased visual acuity, increased intraocular pressure (IOP) indicated by decreased globe compressibility on palpation over closed lid (or pressure > 40 mmHg/ > 5.3 kPa if tonometry available ◈), proptosis
- Secondary: afferent papillary defect, decreased extra ocular movement, eye pain, cherry red macula, optic nerve pallor

Contraindications

- Globe rupture

Preparation

Consent

Risks include bleeding, local infection, and mechanical injury. Benefits include symptom relief and prevention of ocular complications. Alternatives include serial examinations or formal surgical intervention

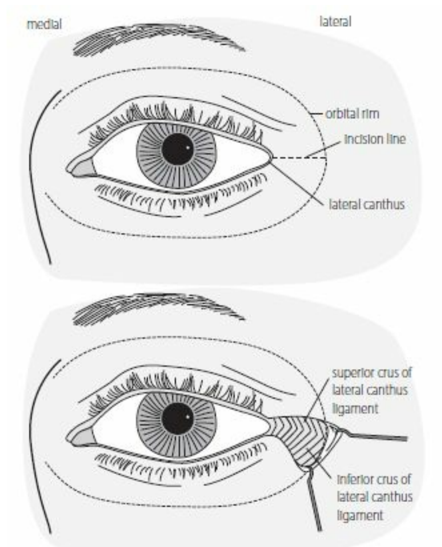
Equipment

- 25 or 27G needle, syringe
- Local anaesthetic with adrenaline
- Haemostat
- Blunt tipped (iris or Steven's) scissors
- Forceps

Procedure

1. Clean skin lateral to eye.
2. Infiltrate anaesthetic into skin at lateral canthus.
3. Clamp lateral canthus (one jaw between globe and canthal tendon, other over skin) to the bony rim of lateral orbit for 1– 2 minutes.
4. Remove clamp and cut along clamp line laterally to orbital rim (1– 2 cm).
5. Retract lower lid down and laterally to expose inferior crus of tendon.
6. Cut inferior crus of tendon to release OCS.
7. Reassess vision, ocular movement and IOP. If IOP still elevated, retract superior lid to expose superior crus, cut to release OCS.

Figure 319.1 Lateral canthotomy



Aftercare

Reassess IOP, visual acuity and extra ocular movement. Incisions will heal without suture or significant scarring.

Potential complications

See consent above.

Interpretation of results

Return of vision or extra ocular movement indicates adequate release of OCS and return of retinal artery perfusion.

Documentation

Indications and exam findings before and after procedure.

Disposition

Emergency ophthalmologic consultation.

320 Thoracentesis

Indications and contraindications

Indications

- Diagnostic: evaluate the cause of a pleural effusion
- Therapeutic: relieve dyspnoea and/or haemodynamic compromise

Contraindications

Relative

- Thrombocytopaenia < 20 000
- Coagulopathy
- Uncontrolled cough or hiccups
- Patients on mechanical ventilation
- Cellulitis or herpes zoster

Preparation

Consent

Risks include PTX, HTX, bleeding, empyema, lung laceration, intra-abdominal viscera injury, subcutaneous emphysema, air embolism. Benefits include symptomatic relief, and facilitation of diagnosis. Alternatives include empiric therapy, chest tube, and surgical intervention.

Equipment

- Sterile gloves
- 3-way stopcock
- Blood transfer set
- 18–20G 50 mm angiocatheter (14 Fr cannula can be used instead)
- 4 × 4 gauze pads, 5 ml syringe with 25–27G
- 15 mm needle and 22G needle
- Local anaesthetic
- Betadine
- Haemostat
- Evacuated containers
- Two specimen containers
- Sterile drapes

Procedure

1. Sitting position with arms and head resting supported on a bedside adjustable table. If unable to sit, the patient should lie at the edge of the bed on the affected side with the ipsilateral arm over the head. Monitor VS throughout the procedure.
2. Insert needle at the posterolateral aspect of the back above the diaphragm, but under the fluid level.
3. Confirm site by counting the ribs based on CXR and percussing out the fluid level. Mark on the skin the location of the fluid level. US ◇ can be used to identify fluid level.
4. Anaesthetise skin, superior surface of the rib and pleura.
5. While exerting steady pressure on the patient's back with the non-dominant hand, insert the thoracentesis needle through the anaesthetised area. Advance the needle until it encounters the superior aspect of the rib. Continue advancing the needle over the top of the rib and through the pleura, maintaining constant gentle suction on the syringe.
6. Attach the 3-way stopcock and tubing, and aspirate the amount needed. Turn the stopcock and evacuate the fluid through the tubing.
7. Remove the necessary amount of pleural fluid (usually 100 ml for diagnostic studies); not more than 500–1 500 ml of fluid at any one time – increased risk of pleural oedema or hypotension.
8. When finished draining fluid, ask the patient take a deep breath and hum, and gently remove the needle. This manoeuvre increases intrathoracic pressure and decreases the chance of PTX.
9. Cover the insertion site with a sterile occlusive dressing.

Aftercare

CXR to exclude PTX.

Potential complications

See consent (above).

Interpretation of results

To determine whether the fluid is an *exudate* or *transudate*, fluid must be sent to the lab for cytology, microbiology, cell count and differential, protein, and LDH. Use Light's criteria (see 🕮 Pleural effusion, p. 634).

Documentation

Document procedure and response, amount of fluid withdrawn, results, and complications.

Disposition

Admit as per the underlying disease. If discharging, return for fevers, bleeding, or difficulty breathing.

321 Chest tube insertion

Indications and contraindications

Indications

Pneumothorax (PTX), haemothorax (HTX), empyema, large pleural effusion.

Contraindications

Relative: coagulopathy.

Preparation

Consent

Risks include PTX, HTX, infection, injury to parenchyma and vessels and re-expansion pulmonary oedema or hypotension. Benefits include symptom relief, restoration of oxygenation and avoidance of surgical complications. Alternatives include empiric treatment, surgical management, and thoracostomy.

Equipment

- Antiseptic solution, sterile drapes
- Medications for pain and sedation
- Local anaesthetic with adrenaline
- 10 ml–20 ml syringe and needles (for local anaesthesia)
- Scalpel
- Large curved forceps
- Chest tube (see below for size)
- Large, non-absorbable suture (e.g. 2–0 silk or nylon)
- Needle holder
- Straight scissors
- Chest tube size by indication:
  - » PTX: stable: 16 Fr–22 Fr; unstable or ventilated patient: 24 Fr–28 Fr
  - » HTX: 28 Fr–36 Fr
  - » Empyema: 20 Fr or larger
- Drainage/tubing:
  - » Any available drainage apparatus with wall suction
  - » Container and sterile water (for water seal) or single self-contained units
  - » Sterile large bore tubing
- Dressing:
  - » Petroleum gauze, 4 × 4 inch sterile gauze
  - » Wide elastic tape or other adhesive dressing

Procedure

1. Positioning: supine, or Fowler’s position. Ipsilateral arm above head. Find insertion site: 4th–5th intercostal space,

just anterior to mid-axillary line.
2. Preparation: scrub the chest wall with antiseptic solution and sterilely drape allowing visualisation of landmarks.
3. Estimate chest tube depth: lay the tube along the chest wall, with the tip reaching the lung apex. Note the location on the tube where it is expected to exit the body.
4. Anaesthesia: sterile procedure, raise a skin wheal at the area of incision, down to the rib and periosteum. The needle should travel above the rib to pierce the pleura. Upon aspirating air, blood, or fluid, place anaesthetic along the pleura.
5. Incision: 3 cm, through skin and subcutaneous tissue, parallel to the rib.
6. Tube placement: use a curved clamp to bluntly dissect to the underlying rib. Run the clamp over the top edge of rib and gently push the clamp through the pleura. Hold the clamp in the palm of your hand, with your index finger 2–3 cm from the tip to allow it to just penetrate the pleura.
7. Once in the thoracic cavity, spread the clamp open. Close the clamp and place the index finger of your non-dominant hand next to the clamp within the thorax. Remove the clamp. Sweep finger around the cavity to ensure the lung is not attached to the pleura. Caution: rib fractures can cause lacerations. Place the tube, alone or loaded onto a curved clamp, into the pleural space to the depth previously confirmed. Ensure the last drainage hole is intrathoracic. Direct the tube along the thoracic wall in an apical-posterior direction. If placed correctly, the tube should spin easily, have fogging with respiration, or tidaling of blood or fluid with respiration. Attach tube to drainage system.
8. Secure the tube: make a horizontal mattress suture encircling the tube (leave both ends equally long). Tie the loose ends with a single surgeon’s knot at the skin. Tie the remaining long loose ends around the tube.

Aftercare

- Dressing:
  - » Wrap petroleum gauze around the tube as it exits the skin, then wrap a cut 4 × 4 gauze around the ICD
  - » Place elastic tape over the dressing, and wrap around the ICD at the skin
  - » Tape the ICD to the skin distally
- Confirm placement:
  - » CXR

Potential complications

See consent, above.

Critical documentation

Document side of placement, medications used, sterile technique, number of attempts, findings on entering the pleura, size placed and any complications.

Disposition

Admit until the tube is removed.

322 Emergency thoracotomy

Emergency thoracotomy delivers rapid thoracic cavity access to relieve cardiac tamponade, control haemorrhage, provide open cardiac massage, and control systemic air emboli. It has very low survival rates, requires immediate availability of operating theatre, and a full surgical team, and haemotransfusion capability.

Indications and contraindications

Indications

- Absolute: cardiac arrest after penetrating chest injury where there was initial, witnessed signs of life
- Relative: consider for cardiac arrest in the resus room after blunt chest trauma. Survival rates extremely poor

Contraindications

- Trauma arrest in pre-hospital setting
- Penetrating trauma with CPR >15 min and no signs of life
- Massive non-survivable injuries

Preparation

Consent

Emergency procedure, consent is implied.

Equipment

- Eye/face protection, gloves
- Scalpel (#10 or #20 blade)
- Heavy scissors
- Large clamp
- Light source
- High volume suction
- Laparotomy sponges
- Rib spreader (Finochietto retractor)
- Long tissue forceps – smooth and toothed
- Long and short needle holders
- Gigli saw
- Aortic clamp
- Suture scissors and material (3-0 or larger silk on large curved needle)
- Haemostats
- Balloon catheter (Foley, 5 ml balloon)
- Sterile towels and drapes
- Skin stapler
- Teflon patches
- Internal defibrillator and paddles

Procedure

1. Intubate patient and place bilateral needle thorocostomies to relieve presumed tension PTX. Establish IV access for transfusion.
2. Position patient supine, left arm abducted (left thoracotomy to access the heart is standard, may need to extend to right side).
3. Apply antiseptic solution over entire chest and bilateral axillae.
4. Make a deep incision to intercostals with scalpel from the left sternal margin (below nipple line), extending arc to posterior axillary line following the interspace.
5. Cut intercostal muscles with heavy scissors along incision.
6. Insert rib spreader and open retractor fully, displace lung to locate heart.
7. Perform pericardiotomy: 'tent' the pericardium with forceps at most anterior point to avoid phrenic nerve, incise pericardium with scissors and extend opening longitudinally with finger to relieve tamponade and deliver heart from pericardium.
8. Temporise cardiac injuries: apply direct pressure on small wounds. A Foley catheter may be inserted through a cardiac wound, the balloon inflated and gentle traction applied to limit haemorrhage until definitive operative repair. Staples can provide closure of ventricular wounds. Clamp atrial wounds. Definitive repair should be performed by surgical team in operating theatre.
9. If no apparent left thoracic haemorrhage: extend incision across sternum. Access right hemithorax with technique described above. Cut through sternum with shears, scissors or Gigli saw.
10. Clamp haemorrhaging intercostal or hilar vessels. May twist hilum to control pulmonary haemorrhage or air

embolus.
11. Clamp descending aorta to redistribute volume and control extrathoracic haemorrhage.
12. Perform two-handed open cardiac compressions.

Aftercare

If perfusion is restored, provide anaesthesia; continue haemotransfusion.

Potential complications

• Neurologic impairment, death, organ injury, phrenic nerve injury
• Multiorgan failure
• High risk to providers from body fluid exposure/fractured ribs

Documentation

Indications, signs of life on arrival, blood products transfused, disposition.

Disposition

Transfer to OT for definitive repair if perfusion is restored.

323 Pericardiocentesis

Pericardiocentesis is percutaneous needle aspiration of fluid from within the pericardial sac.

Indications and contraindications

Indications

• Diagnostic: to help distinguish whether a pericardial fluid collection is due to infection, cancer, or an autoimmune condition
• Therapeutic: cardiac tamponade, pericardial effusions larger than 250 ml

Preparation

Consent

Risks include failure, PTX, HTX, pneumopericardium, arrhythmia, coronary artery puncture, and cardiac arrest. Benefits include restoration of perfusion, symptomatic relief, and accurate diagnosis. Alternatives include empiric therapy, and surgical intervention.

Equipment

• Standard prep and drape
• Local anaesthesia with adrenaline
• 2.75 inch (70 mm) spinal needle (alternatively use CVP set)
• 20 ml, 50 ml syringe

Optional

• ECG monitor ◇
• US ◈
• CVP cannula and 3-way tap is an alternative (leave the cannula in situ for repeat aspirations)

Procedure

1. Consider sedation.
2. Prep and drape the subxiphoid/parasternal area.
3. Infiltrate local anaesthesia.
4. Attach spinal needle to 20 ml syringe.
5. Position patient in semi recumbent position at 45°.
6. Insert to the immediate left of the xiphoid (left subxiphoid angle).
7. Advance the needle towards the left shoulder at a 15–30° angle aspirating as you enter, you may feel a ‘pop’ as you puncture the pericardium.
8. In non-traumatic tamponade, a yellow serosanginous fluid can be aspirated, with improvement of VS and in traumatic tamponade, aspirating about 10 ml of fluid will result in improvement of VSs.
9. Remove needle and dress puncture wound.
10. CXR: to evaluate for complications.
11. Reassess VS; admit for definitive care.

If using an ECG

- Sudden ST elevation on ECG suggests needle contact with myocardium
- Withdraw needle slightly if ST elevation occurs
- ST elevation that persists should prompt complete needle removal

If using US guidance

With probe in the subxiphoid position:
- Needle is inserted adjacent to US probe
- Angle the needle at 45° and directed towards the left shoulder
- Aspirate while inserting needle
- Watch the needle enter the largest pocket of fluid
- Aspirate pericardial effusion

With probe in the parasternal position:
- Position US lateral to needle entry site
- Insert needle perpendicular to chest (90°), 5th intercostal space or immediately lateral to sternum
- Aspirate while inserting needle
- Watch the needle enter the largest pocket of fluid. Aspirate pericardial effusion

Aftercare

Potential complications

See consent above.

Documentation

Record indications, technique, findings, complications.

Disposition

This is a temporising measure; admit for further care and possible pericardial window.

324 Transcutaneous pacing

Also called external pacing, this is a temporary means of pacing a patient’s heart by delivering pulses of electric current through the patient’s chest.

Indications and contraindications

Indications

- Bradycardia (< 60 bpm) with signs of shock (hypotension, altered mental status, chest pain) (* Not all instances of bradycardia require transcutaneous pacing)
- Temporary cardiac pacing until the more permanent transvenous pacing (where available) can be initiated, or until an underlying cause of the bradydysrhythmias can be corrected
- Mobitz II or 3rd degree AV block with symptomatic bradycardia, while preparing for transvenous pacing
- Overdrive pacing of some forms of ventricular tachycardia

Contraindications

- Prosthetic tricuspid valve
- Severe hypothermia
- Prolonged bradyasystolic cardiac arrest
- Non-intact skin for the site of the pads or electrode placement.

Preparation

Consent

Emergency procedure, consent is implied.

Equipment

- Resuscitation equipment and medications
- Airway equipment
- A monitor/defibrillator with pacing capabilities
- ECG electrodes for rhythm monitoring
- Pacing electrode pads
- Sedation and/or procedural analgesia medications

Procedure

1. Place electrode pads in the standard or AP position (black on anterior chest, red on posterior chest) and connect to a monitor/defibrillator.
2. Connect ECG leads.
3. Set pacemaker to *demand* mode.
4. Turn pacing rate to > 30 bpm above patient's intrinsic rhythm.
5. Set current to 50–70 mA and start pacing.
6. Increase current until pacing rate captured on monitor (electrical capture) with a corresponding pulse – use femoral pulse (mechanical capture). Electrical capture is characterised by a wide QRS complex with tall, broad T wave on the ECG.
7. If pacing rate is not captured at a current of 120–130 mA, reposition electrodes and repeat the above.
8. Once pacing is captured, set current at about 10% above mechanical capture threshold.
9. Attempt to get the patient to a centre where a transvenous or permanent pacemaker can be placed while awaiting transfer, actively look for and if possible reverse cause of bradycardia.

Aftercare

Potential complications

- Pacing artefact on the ECG and severe muscle twitching may make the determination of electrical capture difficult. It is advisable to use another instrument (e.g. SpO_2 monitor or bedside Doppler) to confirm mechanical capture
- Watch for a change in underlying rhythm

- Periodically check the area where the electrodes are placed for skin burns or tissue damage. Inspection and repositioning as needed can alleviate these problems

Documentation

Indications for pacing, pre-paced rhythm and 12-lead ECG findings, medications given, energy level/settings required, any complications that occurred, patient assessment and outcome, disposition, and the notification of attending physician and family members.

Disposition

Admit to ICU ◇ or ward.

325 Gastrostomy tube replacement

Gastrostomy tubes (G-tubes) are surgically or endoscopically placed feeding catheters in patients who need long-term nutritional supplementation or cannot take food by mouth.

Indications and contraindications

Indications

- Malfunction of G-tube
- Accidental removal of G-tube

Contraindications

If initial G-tube placement is less than three months old, it is possible to create a false tract upon re-insertion. It takes 1–2 weeks for tract to form after initial procedure. Unless there are major barriers, replacement should be avoided in the first two weeks (insert a foley catheter instead).

Avoid replacement if there is any evidence of overlying infection around G-tube site, such as extensive erythema, exudate, or warmth.

Preparation

Consent

Risks include local infection, bleeding, false tract creation, and placement of tube into peritoneal cavity. Benefits include facilitation of feeding. Alternatives include replacing with a NGT tube, or IV fluids.

Equipment

- Gastrostomy tube of appropriate size
- Foley catheter, 1–2 sizes smaller than G-tube
- 1–2 saline flushes
- Surgical lubrication jelly
- If tube is obstructed, Coca-Cola or other proteolytic enzyme
- 15–30 ml of diatrizoate meglumine (if available) if confirmation of tube placement is needed

Procedure

G-tube leak

- Determine whether intra-abdominal aetiology vs tube malfunction
- If tube malfunction, proceed to replacement

G-tube obstruction

- Flush tube with Coca-Cola or if available, a proteolytic enzyme
- If unable to flush, proceed to replacement

G-tube replacement

- If G-tube is still in place, deflate balloon and remove
- If replacement G-tube is not readily available, use a Foley or other similarly sized catheter
- Replacement should be done as soon as possible so that the tract does not close
- Lubricate tip of catheter and holding close to tip, apply firm pressure to insert into tract
- Never force the tube, as a false tract or separation of the stomach from the external stoma can occur
- Insert G-tube to hub; if using alternative catheter, insert to appropriate depth to reach stomach
- Fill catheter balloon with 5–10 ml (or amount on catheter instructions) of saline or sterile water
- If using alternative catheter, remove and repeat steps above when G-tube becomes available
- Indications for confirmation of the G-tube placement: the track is relatively new (< 1 month old), the tube is difficult to replace, or there is any doubt that the tube is in the stomach
- To check placement, insufflate 20 ml of air and listen for borborygmi over the stomach with a stethoscope. Gastric contents should also be aspirated easily
- If available, tube placement can be confirmed by instilling a small amount of water soluble contrast solution into the tube (15–30 ml) and obtaining two radiographic views to observe the tube and dye in the stomach

Aftercare

Potential complications

See consent (above).

Documentation

Type of tube used for replacement, confirmation of placement.

Disposition

- Discharge if replacement of G tube is successful
- If unable to replace G-tube, place NGT for feeds/hydration and discharge to follow-up with provider who can replace G-tube
- If unable to replace G-tube and unable to provide adequate hydration, admit for IV hydration

326 Abdominal paracentesis

The insertion of a needle into the peritoneal cavity to drain ascitic fluid.

Indications and contraindications

Indications

- Therapeutic: removal of 5 l or more of fluid to reduce intra-abdominal pressure (if it is causing respiratory compromise or abdominal compartment syndrome).
- Diagnostic: removal of a small amount of fluid for investigations in new-onset ascites or suspected spontaneous bacterial peritonitis

Contraindications

- Acute abdomen

- INR > 2, platelet count < 20
- Pregnancy
- Distended bladder, bowel
- Abdominal wall cellulitis

Preparation

Laboratory tests

If possible, obtain platelet count and PT/PTT studies prior to performing procedure, particularly in patients with liver disease.

Consent

Risks include failure, persistent leak, local infection, haemoperitoneum (rare; due to mesenteric variceal bleeding after removal of > 4 l of ascitic fluid), hollow viscous or blood vessel perforation, hypotension, hyponatremia, and hepatorenal syndrome. Benefits include symptomatic relief and accurate diagnosis. Alternatives include empiric treatment.

Equipment

- Antiseptic swab sticks
- Fenestrated drape
- Local anaesthetic with adrenaline
- 14–18G IV catheter
- 10 ml and 50 ml syringes
- Drainage bag or vacuum container
- Specimen vials or collection bottles
- Gauze
- Adhesive dressing
- US ◇ increases rate of success and decreases risk of complications

Procedure

Positioning

1. Empty the patient's bladder (voluntarily or with a catheter).
2. Position the patient supine.
3. Prepare the skin around the entry site with an antiseptic solution.
   Sites:
   - 2 cm below the umbilicus in the midline (through the linea alba)
   - 5 cm superior and medial to the anterior superior iliac spines on either side
4. Apply a sterile drape to create a sterile field.
5. Use 5 ml syringe and 25 G needle to raise a small lidocaine wheal around the skin entry site.
6. Insert the IV cannula directly perpendicular to the selected skin entry point (using US if available). Slow insertion in increments of 5 mm is preferred (to minimise the risk of inadvertent vascular entry or puncture of the small bowel).
7. Attach a 50 ml syringe and aspirate the ascitic fluid.
8. Fill the specimen vials.
9. Attach a urinary drainage bag to the cannula and monitor drainage.
10. Use adhesive dressing to stabilise the cannula.

Aftercare

Potential complications

See consent (above).

Interpretation of results

Samples with > 250 polymorphonuclear cells/µl or a blood:ascitic fluid pH gradient ≥ 0.1 are considered bacterial peritonitis.

Documentation

Technique, complications, appearance and amount of fluid obtained, laboratory results.

Disposition

As per underlying condition.

327 Diagnostic peritoneal lavage (DPL)

When available and patient condition permits, CT remains the study of choice to characterise intraperitoneal injury, but DPL is faster, can be used in unstable patients, and may be particularly valuable within the first few hours after hollow viscus injury when the sensitivity of CT is poor.

Indications and contraindications

Indications

- Rapid determination of the presence of intraperitoneal haemorrhage
- Suspicion of hollow viscus injury
- Suspicion of diaphragmatic violation

Contraindications

DPL can be performed in virtually all patients. Adjusting the technique and site of performance allows relative contraindications to be overcome.

- Relative: prior abdominal surgery or infections, coagulopathy, obesity, second and third trimester pregnancy

Preparation

Consent

Risks include bleeding, peritonitis, injury to bladder or other abdominal and retroperitoneal structures, and wound infection. Benefits include avoidance of unnecessary surgery, and facilitation of definitive care. Alternatives include surgery, transfer for CT, or empiric management.

Equipment

Local anaesthetic with epinephrine, povidone-iodine or chlorhexidine, sterile drape, scalpel (11 or 15 blade), retractors, towel clamps, DPL catheter (urethral catheter or N-G tube also suitable), 10 ml syringe, 1.0 l warm IV fluid (N/S or R/L) and IV tubing without valve, absorbable suture, needle holder.

Procedure

Open method

1. Decompress urinary bladder (Foley catheter).

2. Decompress stomach (NG tube).
3. Surgically prepare the abdomen – observe sterile technique throughout procedure.
4. Inject local anaesthetic (1% lidocaine with adrenaline) at the midline, just below the umbilicus.
5. Make a 2–4 cm long vertical infraumbilical incision to the rectus fascia.
6. Grasp fascial edges with clamps and elevate and incise (1–2 cm) the fascia down to the peritoneum. Make a tiny peritoneal incision, entering the peritoneal cavity.
7. Insert a catheter into the peritoneal cavity.
8. Direct the catheter downwards into the pelvis.
9. Connect the catheter to a syringe and aspirate.
10. If gross blood (> 5 ml) or bowel content aspirated, organ damage is confirmed. Proceed to appropriate surgical management.
11. If not, infuse 1.0 L of warmed crystalloid (10 ml/kg in a child) into the peritoneum.
12. Gently agitate the abdomen to distribute the fluid throughout the peritoneal cavity and increase mixing with the blood.
13. If the patient is stable, let the fluid remain for a few minutes before placing the crystalloid container on the floor and allowing the peritoneal fluid to drain from the abdomen. Make sure the container is vented to promote flow of the fluid from the abdomen; adequate fluid return is > 30% of infused volume.
14. Send a sample to the laboratory for Gram staining and erythrocyte and leukocyte counts.
15. Close the abdomen in layers, and apply dressing over incision.

Closed method

Similar steps are followed for the closed technique except for a small puncture wound that is made on the abdomen, and a DPL catheter is placed over a wire (Seldinger technique).

The use of prophylactic antibiotics is not supported by evidence but may be given where necessary. Sutures for wound closure are removed after 3–7 days.

Aftercare

Potential complications

See consent (above).

Interpretation of results

- Positive (need for surgical intervention): aspiration of > 5 ml free blood or obvious enteric contents; RBC count >100 000/mm^3, WBC count > 500/mm^3, or positive for food fibres or bacteria
- Negative lavage does not exclude retroperitoneal injuries (pancreatic and duodenal injuries, or diaphragmatic tears)

Documentation

Document the procedure, findings and complications.

Disposition

Admit positive DPL for surgical intervention. If DPL is negative, admit or discharge based on underlying clinical condition.

328 Arthrocentesis

Arthrocentesis of major joints is a simple procedure that can be both diagnostic (cell count, culture) and therapeutic (delivery of steroid and anaesthetic, or drainage of blood/effusion). The most common site is the knee. US guidance can increase procedure accuracy and success for some joints.

Indications and contraindications

Indications

Diagnostic

- Synovial fluid analysis for septic joint or crystal-induced arthritis
- Differentiate between traumatic and inflammatory joint effusion
- Fluid for culture, Gram stain, immunologic studies and cell count (joint infections)

Therapeutic

- Relieve the pain of acute haemarthrosis or a tense effusion and to restore range of motion
- Local instillation of medications (e.g. lidocaine, corticosteroids) in acute or chronic inflammatory arthritis or dislocation
- Drain septic or crystal-laden fluid to relieve intra-articular pressure

Contraindications

- NEVER inject through infection of overlying skin or soft tissue
- Coagulopathy

Preparation

Be familiar with the anatomy of the joint. Ensure no bleeding disorders, use of anticoagulants, history of allergy to therapeutic agents. Document signs of joint inflammation, effusion, range of motion, crepitation, deformity, ligamental instability, concurrent systemic infections, and skin findings.

Consent

Risks include infection, recurrent pain, extra-articular injection, bleeding, hypersensitivity reaction. Benefits include identification of aetiology of joint inflammation to allow appropriate therapy. Alternatives include clinical diagnosis, empiric therapy.

Equipment

- Sterile gloves and drapes
- Iodine and alcohol solution, sterile gauze
- Local or regional anaesthetic
- Sterile 18–25 gauge needle
- 1–50 ml syringe (depending on the joint and amount of effusion)
- Tubes for synovial fluid analysis: haematology tube for cell count and differential, sterile tubes for gram stain, cultures and smears. Heparinised tube for crystal analysis. Cytology bottle (for neoplasm)

Procedure

1. Place in a comfortable position with easy access to affected joint.
2. Expose joint and carefully identify the anatomic landmarks. Mark out the exact injection site with a sterile marker or indentation on the skin.
3. Scrub site with iodine solution then alcohol.
4. Wear sterile gloves and drape prepped skin.
5. Apply local or regional anaesthetic. If ice used, place a bag of ice over the drape (caution: maintain skin sterility) and remove after 5 minutes, then perform procedure immediately. If topical anaesthetic is preferred, spray the solution 150 mm from the skin until the skin becomes frosty; perform the procedure within 30–60 seconds.
6. Insert needle through the skin and advance needle into the joint space.
7. Place fluid in appropriate tube.

8. Withdraw needle and apply pressure to the puncture site with sterile gauze for 3–5 minutes.
9. Apply pressure bandage to avoid recollection of fluid or accumulation of blood underneath the skin.
10. Apply ice and elevate joint for 24–48 hours to reduce swelling and pain. Analgesics (NSAIDS) may be given.

Aftercare

Potential complications

See consent, above.

Interpretation of results

Differential diagnosis of synovial fluid analysis

- Bacterial: infectious arthritis
- Elevated WBC: infectious or inflammatory disorder
- Uric acid crystals: gout
- Pyrophosphate crystals: pseudogout

Documentation

Indication, technique, complications.

Disposition

Discharge patient with advice to avoid excessive use of the affected joint for 24–48 hours and to contact the doctor if there is increased pain, fever, skin redness, swelling, excessive bleeding or discharge at the arthrocentesis site.

329 Burr holes

Emergency burr hole craniotomy (also called trepanning or trephining) is the process of making a hole through the skull to treat expanding intracranial haematoma. It may be useful in improving survival. This procedure can be beneficial in low resource settings where there are no neurosurgeons, and may be done with improvised equipment.

Indications and contraindications

Indications

- GCS ≤ 8 with CT evidence of extra-dural haematoma causing midline shift and unequal pupils
- Acute subdural haematoma with midline shift on CT
- High clinical suspicion (e.g. palpable fracture with ipsilateral fixed pupil) with no CT scan available
- Chronic subdural haematoma

Contraindications

- GCS > 8
- Neurosurgical intervention available within a reasonable time frame

Preparation

Consent

Risks include bleeding, infection, seizures, intracerebral haemorrhage, subdural empyema, changes in memory (or behaviour, thinking or speech), paralysis or weakness, cerebral damage, bleeding and cardiac arrest. Benefits include prevention of permanent neurological sequelae. Alternatives include mannitol and symptomatic management.

Equipment

- Scalpel
- Self-retaining retractor
- Hand-held drill and specific drill bits (can use household drill in extremely low resource settings)
- Swab
- Sharp and blunt hook

Procedure

1. Ensure patient is supine and physiologically optimised (intubated, $ETCO_2$ 4.5 kPa, normotensive, c-spine protection, mannitol/hypertonic saline as directed by neurosurgeon).
2. With CT: confirm position of haematoma (most commonly temporal). Count down the number of slices from the top (and multiply by slice thickness) to the centre of the haematoma to calculate how many centimetres below the vertex the burr hole should be.

Without CT: two finger breadths above the ear and two finger breadths anterior to external auditory canal for temporal burr hole.

3. Shave a strip of approximately 5 cm of hair. Mark a 3 cm line of incision.
4. Clean the area with betadine/chlorhexidine.
5. Make an incision straight down to bone. Bleeding (from the superficial temporal artery) can be controlled with direct pressure while continuing the procedure.
6. Push the periosteum off the bone with knife/swab.
7. Insert self-retaining retractor.
8. Push down firmly with drill and start drilling, keeping drill perpendicular to the skull. Ensure an assistant is holding the head still and ideally apply saline wash as you drill.
9. Do not stop, as this will disengage the clutch mechanism, which can be difficult to re-engage manually.
10. Drill until the drill stops spinning. Remove drill.
11. Use blunt hook to remove remaining bone fragments.
12. Extradural blood should now escape.
    - If the blood is subdural, carefully open the dura using a sharp hook to tent-up the dura, and use a new sharp knife to incise the dura in a cruciate manner. Subdural blood is likely to be clotted and more difficult to extrude than extradural
    - If fresh blood is continuing to ooze from the wound, do not try to tamponade. Leaving the self-retainer in place may stop the bleeding. Try to diathermy the skin edges; if not available apply direct pressure to wound edges during transfer

Aftercare

Potential complications

See consent, above.

Documentation

Document the procedure performed thoroughly, the site of the haematoma and the volume of fluid aspirated.

Disposition

Admit – to neurosurgeon ⟐ where available.

330 Escharotomy

Indications and contraindications

Indications

Deep partial thickness and full thickness **circumferential** burns of the chest and or limbs.

Preparation

Consent

Emergency procedure, consent is implied.

Equipment

- Sterile gauze packs, drapes, cleaning solution
- 10 ml syringe with needle
- Local anaesthetic agent with adrenaline
- Scalpel
- Ligatures or diathermy

Procedure

- Anaesthesia is not necessary; use local anaesthesia in unburned skin
- The patient should be receiving IV analgesia already
- Full thickness incision **into subcutaneous fat** using a size 15 blade
- Running a finger along the incision will detect residual restrictive areas
- Incisions must extend above and below into unburned tissue
- Have ligatures available for haemorrhage control, diathermy if available
- Sterile procedure with adequate drapes
- Dress wounds as you would the rest of the burn

Landmarks

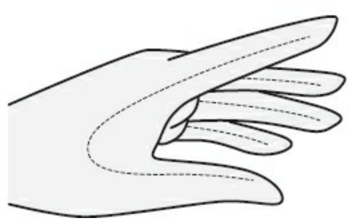


Figure 330.1 Landmarks on the hand

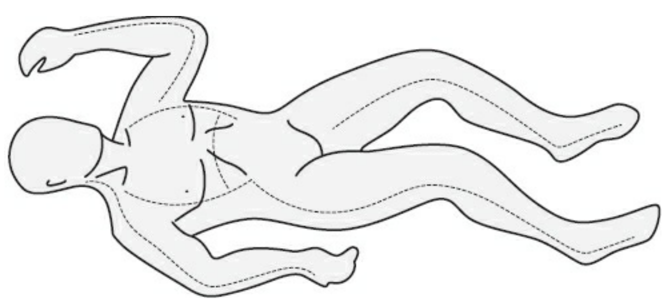


Figure 330.2 Landmarks on the body

Chest and abdomen

- The incision runs longitudinally along the anterior axillary line to the costal margin or the upper abdomen. Perform on both sides

- Connect the two lateral incisions by horizontal incisions. These are convex upwards across the upper chest below the clavicles and across the upper part of the abdomen below the costal margins

Upper limb

- The limb must be in a supine position
- Incise medially and laterally between flexor and extensor compartments
- Be sure to incise anterior to the medial epicondyle
- Avoid transverse incisions along joint creases
- Medial incision passes along medial border of hand to base of little finger
- The lateral incision comes down to the base of the thumb

Lower limb

- Incise medially and laterally between flexor and extensor compartments
- The medial incision passes behind the medial malleolus avoiding the long saphenous vein and saphenous nerve
- The lateral incision must avoid the common peroneal nerve where it crosses the neck of the fibula

Aftercare

Potential complications

Bleeding, local infection, pain.

Documentation

Document medications and technique, and any complications.

Disposition

As for the burn injury.

331 Plaster cast immobilisation

Splints are non-circumferential to allow swelling of the extremity without a significant increase in tissue pressure. Splints can be applied temporarily using basic materials like branches, boards, layers of cardboard, or foam, wrapped with materials such as bandannas or torn shirts or other pieces of clothing.

Indications and contraindications

Indications

Temporary immobilisation to improve pain and discomfort, decrease blood loss, reduce the risk for fat emboli and minimise the potential for further neurovascular injury associated with:

- Fractures/sprains/reduced dislocations
- Tendon lacerations/deep lacerations across joints
- Painful joints

Contraindications

If rapid access to definitive care cannot be assured, open fractures, impending compartment syndrome, neurovascular compromise and dystrophy.

Preparation

Adequate knowledge of the relevant anatomy and potential complications. Reduce angulations in fractures; assess neurovascular status.

Consent

Risks include ischaemia, heat injury, pressure sores, skin breakdown, infection, dermatitis, neurologic injury, compartment syndrome, joint stiffness and muscle atrophy. Benefits include symptom relief and facilitation of healing. Alternatives include formal immobilisation in a cast, and systemic analgesia.

Equipment

- Plaster rolls/sheets or prefabricated splint rolls (Ortho-Glass)
- Stockinette
- Cast padding
- Elastic bandages
- Adhesive tape
- Scissors
- Bucket
- Sheets or pads to protect patient clothing
- Gloves

Procedure

1. Inspect, clean, repair and dress any wounds.
2. Put limb in the anatomical position.
3. Cover patient with sheet or gown to protect clothing.
4. Apply stockinette and roll about three layers of cast padding evenly, smoothly and loosely over the area to be splinted.
5. Place extra padding over bony prominences (prevents pressure sores).
6. Prepare the plaster splint material or Ortho-Glass.
7. Lay the dry splint next to the area to be splinted. Estimate the length and make about 8–12 layers: 8–10 layers for upper extremities and 12–15 for lower extremities. More layers may be needed for large patients.
8. Cut the splint material to the desired length.
9. Submerge the dry material in the bucket of cool water until bubbling stops.
10. Remove material and gently squeeze out excess water until plaster is sloppy.
11. Smooth out the splint to remove any wrinkles and laminate all layers.
12. Place the splint over the padding and smooth it onto the extremity to include the joint above and joint below but not circumferential.
13. Alternatively, place 2–3 layers of padding directly over the wet plaster and apply splint.
14. Fold back edges of stockinette and padding over the ends of the splint.
15. Place the extremity in the desired position and mould the splint to the contour of the extremity using your palms (fingers make indentations in the plaster which causes pressure sores).
16. Secure splint with an elastic bandage.
17. Reassess for vascular compromise, discomfort, or pressure points.
18. Apply adhesive tape to ends of the bandage.
19. Provide sling or crutches as needed.
20. Discharge patient with instructions to elevate limb to reduce pain and swelling. Encourage patient to keep splint dry and teach to recognise signs of infection and vascular compromise.

Aftercare

Pulses and sensation should be checked regularly. If patient complains of tightness, tingling, or numbness, the splint should be rewrapped.

Potential complications

See consent (above).

Documentation

Document all findings as well as the procedure.

Disposition

As per the underlying condition.

Chapter 331: Splinting

332 Tracheostomy tube replacement

Tracheostomy tubes are placed for chronic respiratory failure, anatomic dysfunction, to minimise aspiration risk and to reduce risk of tracheomalacia from long-term intubation. Can be placed open or percutaneously.

Indications and contraindications

Indications

- Cuff malfunction
- Occlusion
- Accidental dislodgement

Contraindications

Infection is not a contraindication.

- Emergency tracheostomy replacement: fresh tracheostomy tract (< 7 days for percutaneous; < 3 days for open)
- Routine tracheostomy replacement: respiratory distress, haemodynamic instability, or known/suspected complications of passing a tracheostomy tube

Preparation

Consent

Risks include failure, insertion into a false tract, aspiration, hypoxia, bronchospasm, haemorrhage, and cardiac arrest. Benefits include airway protection, and prevention of aspiration. Alternatives include observation, and intubation.

Equipment

- Suction
- Intubation equipment: laryngoscope, O_2, ET tubes
- Tracheostomy tubes can be made of stainless steel or plastic. The tube is defined by the size of the inner diameter, outer diameter, length and curvature. Tracheostomy tubes can be cuffed, fenestrated, single or double lumen

Procedure

1. Wash hands, use gloves and eye protection.
2. Identify the size and style of the tracheostomy and collect one of the same size and a size smaller. Verify that the components fit together and that cuff inflates. Age-appropriate endotracheal tube can be used temporarily if needed.
3. Insert the obturator into the new tracheostomy tube and lubricate the tube using a water-soluble lubricant. For the first time tracheostomy exchange, verify that resources are available for difficulty passing the tube (anaesthesia, surgeon, etc.).
4. Place the patient in the supine position with a shoulder roll/pillow under their shoulders to facilitate extension of

their neck.

5. If possible, preoxygenate the patient with O_2 or hand bagging.
6. Suction tracheostomy and airway gently, using soft-tipped suction catheter. Small amounts of normal saline can be used to break up thick secretions if needed.
7. Unhook the flanges, remove the dressing and clean around the stoma.
8. Deflate cuff and remove original tracheostomy tube following the curvature of the tube. Alternatively, suction catheter, silicon catheter or similar device can be placed through the original tracheostomy and used as a guidewire in a modified Seldinger technique.
9. Use a tracheostomy hook to hold the stoma open (optional but recommended in small stomas).
10. Quickly insert the new tracheostomy tube with the obturator in place in the reverse, singular motion following the curvature of the tube. STOP if resistance is met, as this can represent creating false passage.
11. Once the flanges are flush against the patient's neck, remove the obturator, quickly. O_2 exchange cannot occur while the obturator is in place.
12. Insert the inner cannula and attach the tracheostomy to the ventilator, bag valve mask or tracheostomy collar. Reinflate the cuff and check the cuff pressure with a manometer, if available (optimally between 15–30 cm H_2O). Low pressure can result in hypoventilation; high pressure can result in tracheal mucosal damage. If no manometer available, use minimal occlusion volume technique: listen to the neck just below the thyroid cartilage with a stethoscope and insert 0.5 ml of air into the cuff. Once no leak can be auscultated, note the volume inserted.
13. Tie the tracheostomy tube back into place using the flanges.

Failed recannulisation

• If you cannot insert the tube, attempt to insert a smaller tube
• If also unsuccessful, emergent intubation may be required to secure airway

Aftercare

Potential complications

See consent (above).

Documentation

Date of initial tube placement, indication for tube change, size of replacement tube, complications.

Disposition

Admit per underlying condition.

333 Cricothyrotomy

A life-saving procedure used to establish an emergency airway when a patient cannot be oxygenated/ventilated by other means. In adults, surgical cricothyrotomy is the preferred method.

Indications and contraindications

Indications

• Inability to oxygenate and ventilate patient non-invasively
• Failed intubation attempts
• Massive facial trauma
• Upper airway obstruction (oedema or foreign body)

Contraindications

Absolute

- < 8 years old, *surgical* cricothyrotomy is contraindicated – use *needle* cricothyrotomy

Relative

- Expanding haematoma in the neck

Preparation

Consent

Emergency procedure; consent is implied.

Anatomy

The cricothyroid membrane is located between the thyroid cartilage and the cricoid cartilage. Palpate the laryngeal prominence of the thyroid cartilage and move inferiorly until you feel a shallow depression.

Equipment

Needle cricothyrotomy

- 14 g IV catheter with needle
- 5 ml syringe
- Endotracheal tube connector
- High pressure wall O_2 source, y-connector or connecting tubing with a side hole OR BVM device

Traditional surgical cricothyrotomy

- Antiseptic solution
- Scalpel
- Tracheal hook
- Trousseau dilator or small forceps
- 6.5 endotracheal or tracheostomy tube

Procedure

- Wear face mask and eye protection
- Position patient with neck hyperextended (contraindicated if cervical fracture suspected)
- Apply antiseptic solution to anterior neck

Surgical cricothyrotomy

- Make a horizontal incision through the cricothyroid membrane
- Grasp the superior part of the membrane with tracheal hook and apply gentle upward traction
- Insert the Trousseau dilator (or small forceps) and, with the blades horizontal, expand the incision vertically
- Rotate Trousseau dilator 90° and re-open blades
- Insert endotracheal or tracheostomy tube and inflate cuff
- Secure tube in place using a tracheostomy tie or suture
- Attach the tube to the ventilation device
- Confirm placement by listening to lungs and looking for chest rise

Needle cricothyrotomy

- Attach a 14 g IV catheter with needle to a 3 cc syringe filled with 1 ml of saline
- Locate the cricothyroid membrane and insert 14 gauge catheter with needle at a 45° angle towards feet while

aspirating with the syringe
- Once the membrane is pierced, bubbles in syringe will indicate entry into the airway
- Steady the needle and syringe and advance the catheter into the airway then remove the needle
- Secure the catheter to the neck using tape or suture
- Attach the syringe, with the stopper removed, to the catheter
- Once the cannula is in position, connect to high-flow O_2 using a y-connector or tubing with a side-hole and commence jet insufflation
- Connect manual jet insufflation (percutaneous transtracheal jet ventilation) device to high flow O_2 at 10–15 l/min. Where a commercial device is not available it may be possible to connect the O_2 tubing directly into a 5 ml syringe with the plunger removed and a hole cut in the O_2tubing. Manually ventilate by closing the circuit for 1 second and releasing for 2–4 seconds

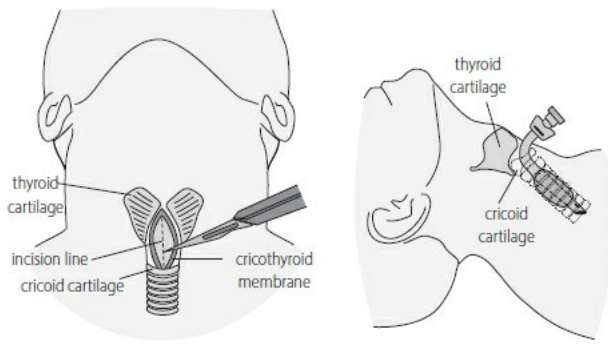


Figure 333.1 Surgical cricothyrotomy

Potential complications

Early:
- Bleeding
- Injury to surrounding structures
- Unsuccessful placement
- PTX/pneumomediastinum
- Subcutaneous emphysema
- Oesophageal perforation

Late:
- Infection
- Voice changes
- Subglottic or glottic stenosis
- Tracheomalacia
- Tracheoesophageal fistula

Documentation

Document the procedure, indications, any complications, and clinical status following the procedure.

Disposition

Admit to ICU ⟐.

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Part

7

A. Process

334 Ethical considerations in resuscitation

Decisions about resuscitation depend on the patient's medical condition, patient wishes, family concerns, cultural issues, and local laws. The goal of resuscitation is restoration of physiological function and quality of life.

Decision-making in resuscitation

All health establishments should have a policy about initiation and termination of CPR; this must be communicated widely and understood by all relevant staff.

- Decisions about CPR are ultimately made on a case-by-case basis
- Advance discussion and planning is important for those at risk of cardiorespiratory arrest
- Where no explicit advance decision has been made, presumption should be in favour of initiating CPR
- If the patient lacks capacity, family should be involved in planning discussions.
- If a patient with capacity refuses CPR, or a patient lacking capacity has a valid and applicable advance decision refusing CPR, this should be respected

'Do Not Resuscitate' orders

DNR orders are made to avoid unnecessary resuscitation, ideally planned in advance with full involvement of the patient and relatives. A DNR order does not override clinical judgement in the event of an unexpected reversible cause of arrest. DNR orders address resuscitation and do not constrain other aspects of treatment.

Medical futility

Determination of medical futility is not an ethical judgement about quality of life, but implies that available medical intervention will not increase likelihood of survival. It is a medical determination made by providers; patients and families do not determine medical futility. The relative availability of clinical resources will impact decisions about

futility, presenting a recurrent dilemma to providers, as conditions that might be survivable in one location may not be survivable elsewhere.

335 Palliative care: definition and principles

WHO defines palliative care as an approach that improves the quality of life of patients and families facing the problems associated with life-threatening illness. Palliative care involves the prevention and relief of suffering by means of early identification and appropriate treatment of pain and other physical, psychosocial and spiritual discomfort. Palliative care is holistic in nature and aims to care for the whole person. WHO defines palliative care for children as the active total care of body, mind, and spirit, (and care of the child involves supporting the family).

Principles of palliative care

Provision of palliative care is relevant from the point of diagnosis until death, and beyond (as bereavement support for survivors). Palliative care includes, but is not limited to, end-of-life care (with which it is often confused). It requires a multidisciplinary approach that engages family and available community resources. WHO outline principles that underpin palliative care:

- Provides relief from pain and other distressing symptoms
- Affirms life and regards dying as a normal process
- Intends neither to hasten nor postpone death
- Integrates the psychological and spiritual aspects of patient care
- Offers a support system to help patients live as actively as possible until death
- Offers a support system to help the family cope during the patient's illness and in their own bereavement
- Uses a team approach to address the needs of patients and their families, including bereavement counselling, if indicated
- May enhance quality of life and positively influence the course of illness
- Is applicable early in the course of illness, in conjunction with other therapies that are intended to prolong life, such as chemotherapy or radiation therapy, and includes those investigations needed to better understand and manage distressing clinical complications

The need for palliative care

Palliative care plays a key role in alleviating the pain and symptoms for patients with communicable and non-communicable diseases. More than 60% of the attributed 57 million annual deaths could benefit significantly from palliative care; up to 60% of patients experience moderate to severe pain and most of them receive inadequate treatment. Provision of palliative care is a major public health issue.

336 Patients with HIV infection in the acute care setting

Screening

In areas where HIV reaches a certain prevalence (see www.WHO.int and www.CDC.gov for specific criteria), routine HIV screening is recommended for all acute care patients with unknown status. Early diagnosis enables optimal care, gives access to interventions, and facilitates appropriate healthcare and social welfare planning.

Symptomatic patients

In patients with symptoms or signs of possible HIV infection, including unconscious or altered patients, testing is part of routine clinical management.

Asymptomatic patients

We recommend voluntary HIV testing and counselling in all high prevalence areas.

Procedure

Pre-test discussion should include:

- Clinical and prevention benefits of HIV testing
- Importance of making an informed decision about testing
- Confidentiality of the testing and results
- A description of the testing and counselling process
- The right to decline HIV testing

Post-test

Results should be given in person by a trained practitioner with full explanation of the treatment, care and support services available. Patients can refuse results.

Typically acute care providers will need to refer the patient for further in-depth counselling.

Key considerations in post-test counselling:

- Assess the person's readiness to receive result
- Communicate results simply and clearly; give patient time to consider results and ask questions
- Discuss options for partner involvement in HIV testing and counselling
- For negative results, explain how to remain negative. For positive results, explain how to prevent re-infection and transmission of the virus to others, including use of condoms

Key considerations for patients testing positive:

- Explain about referral for further management
- Arrange for referral for further counselling, care, treatment or support services as appropriate, e.g. treatment of opportunistic infections, STI, TB management, PMTCT, family planning, nutrition and psychosocial support

Children

It is important to determine the HIV status of at-risk infants and children at first contact with a health facility. Early diagnosis ensures timely treatment and improves survival. When a mother is known to be HIV positive, the child is considered exposed, but if the mother's status is unknown then parent or guardian consent is required to test the child. When parents and guardians are not available to consent, testing decisions should be based on clinical necessity and provider estimation of the importance of immediate testing to facilitate urgently required care. In situations where testing will not impact immediate care, testing should be delayed to obtain parental consent.

The diagnosis of HIV in a child usually implies that other family members may be living with the virus, though they may not be aware of it. Care of the HIV-infected child should also involve education and support of family. The age at which children may be directly counselled about their own HIV results varies by region, and providers should be aware of local regulations.

337 Approach to suspected child abuse

Identification of abuse

While there is great cultural variation in what is considered appropriate and acceptable discipline for children and equally wide variation in legal statutes regarding determination of child abuse, any child who presents to an acute care facility for an intentional physical or psychological injury should undergo full evaluation, including a home safety evaluation. Child abuse is found in all social and racial groups. It is the clinician's duty to protect the child from further harm: this may be the only opportunity to intervene.

Identify discrepancies between reported history and injury pattern. Longer times between injury and hospital visit should raise suspicion of abuse. Coexistence of new and old injuries or history of hospital visits for injuries should raise concern. In addition, there are specific injury patterns associated with intentional harm.

Suspicious injuries

Head

• Perioral injuries, subdural hematomas, injuries to the eye, skull fractures

Trunk and long bones

See 📖 p. 522 and p. 536 for specific injuries associated with non-accidental trauma.
• Old and new scars
• Burns – especially sock and glove distribution (from forced immersion) or cigarette burns
• Any 'unusual' or patterned wounds
• Perineal and genital injuries
• Leg fractures in non-ambulatory children
• Any long bone fracture in a child < 3 yrs

Reporting

Legal reporting requirements vary from country to country, but reporting is essential to protecting patients from further harm, and clinicians have an ethical imperative to report abuse to authorities. Facilities should clearly identify contact numbers for relevant local authorities (law enforcement, social workers, safe houses, etc).

> There is a difference between an anxious and a frightened child. It is a medical art to separate the one from the other. Always ask for help from a senior doctor or social worker if you are unsure. Approximately 50% of children who die from abuse have been abused before.

338 Domestic and intimate violence victims

Any person, independent of age or gender, who presents to care for physical or psychological trauma due to sexual or non-sexual related abuse by a partner, relative or other close contact, should be treated and protected. Women are disproportionately affected by domestic and intimate partner abuse.

Key historical features

Ask:
• Have you been threatened, frightened, hit or slapped, or felt coerced within the last 12 months?
• Do you feel safe to go home?
• Is there anyone who frightens you?

Signs and symptoms

Look for:
• Unusual injuries (burn marks, rope marks, etc.)
• Injuries to the head (especially eyes), perineum or genitals
• Coexistence of new and old injuries
• History of repeated hospital visits for injuries
• Partners or relatives unwilling to leave patient alone with providers
• History of substance abuse in patient or home environment
• Patient suicidality
• Mentally or physically handicapped patients with injuries

Reporting requirements

Legal reporting requirements vary among countries, but reporting is essential to protect patients from further harm, and clinicians have an ethical duty to report abuse to authorities. Facilities should clearly identify contact numbers for relevant local authorities (police, social workers, safe houses, etc).

The social acceptability of domestic violence is highly variable. According to UNICEF, the percentage of women who think that a male partner is justified in physically abusing her under certain circumstances is, for example: 85% in Guinea, 85% in Zambia, 85% in Sierra Leone, 81% in Laos, and 81% in Ethiopia. Patient or family belief that abuse is acceptable does not mitigate provider responsibility to treat, protect, and report.

339 Sudden infant death syndrome (SIDS)

SIDS is the leading cause of death among infants 1–12 months old in high-income countries. No reliable African data are available. The exact cause is unknown. SIDS is more prevalent in males, but ethnicity and socioeconomic status do not appear to be associated. Typically the infant is found dead after having been put to bed at home, and exhibits no signs of suffering. Some countries use the term 'sudden unexpected death in infancy' (SUDI).

SIDS is a diagnosis of exclusion

- The infant's death must have been sudden and unexpected
- In-depth medical history of infant and family should be reviewed
- An adequate postmortem examination must be performed
- Where available, a standardised SIDS protocol should be followed for evaluation, communication and reporting

Risk factors

Identification of risk factors **may prevent SIDS in siblings**:
- Prone sleeping position
- Sleeping on a soft surface
- Overheated rooms
- Maternal smoking during pregnancy
- Young maternal age
- Preterm birth and/or low birth weight

Exclude homicide or accidental death

SIDS may be explained by child abuse or underlying medical disorders, or by accidental suffocation or entrapment during sleep. Look for:
- Previous apparent life-threatening event in the same household
- Previous unexpected or unexplained death of other infants in the household
- Evidence of current or prior injury in the patient
- Family history of SIDS

Information that should be always collected:
- Time last seen alive
- Time found unresponsive
- Person who found the infant
- Sleeping environment
- Sleeping position when put to sleep/when found
- Medical history (infant and parents)

Responding to a case of SIDS

Despite the fact that they have done nothing wrong, many parents will experience powerful guilt feelings. Providers should:
- Offer a highly professional, compassionate, empathic, supportive and non-accusatory response
- Provide leadership and protection for the family
- Be sensitive to cultural beliefs, values, and practices of the family
- Allow parents time to express their grief and to ask questions

• Be prepared for families to express difficult and overwhelming emotions

Coping with SIDS

Consider staff debriefing as experience with the death of a baby is a traumatic event for all involved.

340 Consent

Consent for clinical care in the acute setting is given in many forms, and emergency conditions must often be managed without explanation, discussion or explicit permission from patients or families. In the case of interventions that must be delivered rapidly to avoid substantial morbidity or mortality, implied consent is usually considered adequate. Some procedures (e.g. IV catheter placement) are considered to involve such limited risk and to be such a common part of basic care, that by convention, explicit consent is rarely sought. In most other cases, patients should provide explicit written or verbal informed consent for all clinical interventions. The term 'informed' consent suggests that patients or their proxies have an understanding of the risks, benefits and alternatives of the proposed intervention.

Elements of informed consent

There are three components to informed consent: threshold, information and consent.

Threshold elements

Competence

Patients (or consenting proxies) must be able comprehend relevant information about the proposed care intervention and be capable of making a reasoned decision based on that information. Competence is a legally-defined capacity in many settings. In most settings, patients must be of a certain age. In general, their ability to understand information and make rational decisions in their on interest must not be compromised by altered level of consciousness or other mental deficit, by the influence of medications, or by coercion in any form.

Voluntariness

A patient's right to accept or refuse medical treatment is fundamental, unless they are incompetent (incapable of making decisions for themselves), or in cases of legislated examinations.

Information elements

Disclosure

Disclosure is the delivery of information about risks, benefits and alternatives. Some countries use a 'reasonable patient' standard: the form and content of disclosure depends on the knowledge and background of the patient, and all commonly occurring risks, as well as rare, serious risks must be disclosed. The more likely a bad outcome is, the greater the obligation to discuss it beforehand.

Some countries use a 'reasonable physician' standard: a patient should receive information that any reasonable physician with the same background and practicing in the same community would disclose in a similar situation.

Recommendation

Based on their professional judgement, clinicians should provide patients with a recommendation (derived from the provider's best estimation of what will most effectively serve patient interests).

Understanding

Providers should confirm that patients/proxies understand and appreciate risks and benefits (often by asking them to describe their understanding of the situation).

Consent elements

Decision

The patient must explicitly decide to accept or refuse treatment.

Authorisation

Authorisation may be verbal or written. Verbal authorisation should be described in the patient record (and witnessed, if the procedure is risky or controversial). Written consent should be ratified by patient or proxy signature on a fully completed and witnessed consent form. Written consent is always preferable.

Consent versus informed consent

Simply telling a patient about a procedure and having them sign a form does not constitute informed consent. Submission to treatment does not constitute consent.

Acting without informed consent

Interventions should not be performed without the full informed consent of the patient, unless the patient is unable to consent and timely intervention is required to avert morbidity or mortality (and there is no prior patient refusal documented). Clinicians have an ethical and legal duty to render emergency care in such situations. Where emergency medical treatment has been provided without consent, patients or their proxies should be fully informed once this is possible. They should also be made aware of their right to refuse further treatment.

341 Medical documentation

A health record is documentation made by a health care professional of clinical consultation, examination or management. Medical documentation, medical record, health record, and clinical or medical chart are used interchangeably.

A clinical chart should document presentation (history and physical examination), all investigations and interventions, and medical decision making. A critical function of the medical chart is to communicate essential information to future providers. There must be sufficient detail for another healthcare professional to seamlessly take over care. A chart can comprise handwritten and/or electronic material, correspondence between clinicians, laboratory reports, imaging records, photographs, videos and other recordings, and printouts from monitoring equipment. Medical records should be retained in non-erasable ink. All content should be expressed in professional language. Personal commentary is not appropriate.

Common documentation failures

Failure to date and sign; inaccurate, illegible, or insufficient information; failure to retain records; storage system that renders records inaccessible to future providers.

Special care is needed when altering or correcting medical records for inaccurate information. For handwritten records, the original information should be marked out with a single line and remain legible. New information should be initialled and dated. For electronic records, the system should be designed to record the time, date, and author of alterations, and to retain all prior versions.

Privacy

Records must be kept securely to avoid damage and unauthorised access; they may be shared with other members of the healthcare team involved in clinical management. Patients have a right to privacy, and any other sharing of the medical record requires explicit patient consent.

Considerations

- Medical notes belong to the institution, but the patient has a right to copies
- Medical documentation can be used as evidence in a court of law

- Your legal defence may only be as good as the quality of your medical documentation
- Documentation of an intervention should be compiled only once the intervention has been performed
- Records should be kept for the duration stipulated by local law
- Digital medical records must be used with appropriate encryption and/or password protection

342 Communication within the acute care team

Effective communication is known to improve patient safety, teamwork and operational efficiency in acute care delivery.

Know the names, roles and responsibilities of your team and **know the leader**. Name tags are useful; introduce yourself to those you don't know. When communicating in a busy situation or with many people, use direct address: call people by name and make eye contact.

Know your role and expectations; ask if unsure.

Resolve conflicts quickly (tension undermines teamwork and patient safety).

Plan for regular team communication. Agree on dedicated times and structure; who should be present; explicit agenda. Ensure that all cadres of the team are represented at team meetings.

Structure process and content of team communication to ensure members have a shared understanding. Mnemonics and checklists can be useful, e.g:

- Process tools: COLD – **C**onnect the patient to monitoring, **O**bserve to make sure they are safe, **L**isten to information, **D**elegate care and activities using a checklist and answer any questions
- Content tools: ISBAR: **I**dentity, **S**ituation, **B**ackground, **A**ssessment, **R**ecommendation

Listen actively. Value the contribution of all team members. Validate the speaker, pay attention, make eye contact and show interest, allow others to finish without interruption. Be aware of your non-verbal communication and avoid multi-tasking as you will probably be distracted from listening or at least give the impression of being distracted.

Repeat back. Restate the main points to confirm that the message you received (your understanding) matches what the sender intended (what they thought they said). Provide an opportunity for questions. Create **feedback loops:** when assigned a task, you should confirm acceptance so that everyone is clear on who has been allocated a task; explicitly confirm when the task has been completed.

Use multiple modes to communicate and reinforce important information in verbal AND written language. For example, **share information visually** in an easily accessible location- visual communication or patient journey boards can effectively transmit a lot of information to many people at a glance.

343 Communication with patients and families

The ability to communicate effectively with patients and families is a fundamental skill for health professionals. Communication in emergency conditions poses numerous challenges that can lead to suboptimal interactions.

Three important domains of communication are:

Content – what is actually being said
- Illness and nature of treatments
- Prognosis for outcomes and quality of life
- Potential complications
- Alternatives to treatment

Process – how it is being said
- Repeat information, as it is difficult to process and retain
- Ensure that senstive information is discussed only in private
- Include written materials when appropriate
- Assess understanding by asking receipient to express what they have understood

• Be sensitive to cultural differences that may impact interpretation

Perceptual – dealing with feelings and emotions
• Be aware of own biases, attitudes and prejudices
• Be aware of own thought processes, emotions, and decision making

The African health context comprises people from many cultures and backgrounds who have diverse understanding and beliefs about healthcare.

Shortage of staff is often given as the reason for failure to communicate explicitly with patients and families; health professionals are responsible for creating time and opportunity to achieve effective communication, as it is an essential part of emergency care. Clear communication will have a direct impact on patients' and families' abilities to follow care recommendations. Patients and families may not feel empowered to ask for information to address their concerns, and providers should always offer to answer any questions.

Effective care includes ensuring that patients and families:
• Are able to speak with the treating doctor or nurse
• Have their questions answered honestly and sensitively
• Feel that hospital staff care about them
• Know of expected outcomes
• Are assured that the best care possible has been given
• Have explanations given in understandable terms
• Are given regular updates on progress
• Are assured of the comfort of their relative
• Are informed about transfer plans while they are being made

344 Cultural considerations in emergency care

Africa is a continent with a wide range of national, religious, cultural, ethnic and racial identities. Illness and our response to it are shaped by all of these factors. Individual beliefs, perceptions and coping skills have a profound effect on the interactions between patients and providers. Failure to appreciate and respond to these forces increases the risk of misdiagnosis, interferes with patients' willingness and ability to follow therapeutic recommendations, and disrupts communication and relationships.

Health care providers should be aware of the impact of social and cultural factors on health beliefs and behaviours and be equipped with the tools to manage these appropriately. Improving cultural awareness will facilitate shared expectations amongst providers and patients.

Understanding the patient

Identifying relevant core cross-cultural issues

• Styles of communication: how does the patient communicate?
• Mistrust: does the patient mistrust the healthcare system?
• Autonomy, authority, and family dynamics: how does the patient make decisions, or who makes decisions for the patient?
• Role of provider: what does the patient expect of us? What is our role?
• Traditions, customs and spirituality: are there collective beliefs that influence the patient?
• Sexuality and gender: how do these factors impact the patient's decision making?

Exploring the meaning of illness for the patient

Patients' ideas about diseases and treatments shape their behaviour; understanding these ideas improves provider

insight into patient behaviour. This is particularly important when patients find it difficult to follow treatment recommendations. Explore the patient's:
• Explanatory model (ideas about illness, its causes and its impact)
• Agenda (goals and desires for the healthcare interaction), and
• Illness behaviour (behavioural accommodation to the illness state)

Determining the social context

Social and cultural factors are deeply intertwined. Consider the patient's:
• Degree of control over personal environment
• Social stressors and support network
• Literacy and language

Negotiation to empower patients in healthcare

Negotiation between patient and provider is essential to an effective therapeutic relationship, and includes the following components:
• Relationship building
• Agenda setting
• Communicating about the assessment
• Clarifying problems
• Agreeing on a shared management plan

Interpreters

Clear linguistic understanding is critical to a successful clinician-patient interaction when the clinician and patient are not able to fluently communicate in a single language. Having a professional staff interpreter is ideal, though rarely possible. Clinical staff members may be able to provide translation, though there are often challenges with balancing other duties. When using family members to interpret, be aware that information and communication will be greatly affected by the familial relationships.

When communicating with a patient via an interpreter, arrange a triangular position—you facing the patient, with the interpreter on the side or behind you. Talk directly to the patient, not the interpreter (in first person, with eye contact). If you wonder about meaning or length of a response, ask the patient and interpreter to clarify. Assist the interpreter by using single questions or short phrasing.

345 Essential equipment and drugs for facility based emergency care

Although available resources vary among facilities, a minimum set of equipment and range of medications is required to be able to provide appropriate emergency care for a range of patient presentations. The lists below are based on the Emergency Medicine Society of South Africa's practice guideline; facilities should work towards compliance with these recommendations.

Devices to open and protect airway	
Laryngoscope set	handle with adult and paediatric blades, spare bulbs and spare batteries
Tracheal tubes	uncuffed (sizes 2.5–5.5 mm)
	cuffed (sizes 3.0–8.5 mm)
Water-soluble lubricant / KY jelly	
10 ml syringe	
Tape or equivalent to tie tube in place	
Oropharyngeal airways	sizes 000 – 5
Nasopharyngeal airways	sizes 3 – 7
Devices to confirm tracheal intubation	
Oesophageal detector device	

End tidal CO2 monitoring	include single use colorimetric devices
Equipment for difficult intubation	
Introducers for ET tubes	adult and paediatric stylets
Magill's forceps	adult and paediatric
Laryngeal masks	sizes 1 – 5
Gum elastic bougie	adult and paediatric
Devices to deliver oxygen and to ventilate patients	
Bag valve ventilation devices	with oxygen reservoir and adult, paediatric and neonatal masks
Oxygen delivery devices	partial rebreather masks, nebuliser masks, nasal prongs and T-piece
Oxygen supply	with flow regulator and oxygen tubing
Equipment to diagnose and treat cardiac dysrhythmias	
ECG monitor defibrillator	with conductive paste or pads, paddles, electrodes and razor
Cardiac arrest board	
Devices to gain intravascular access	
IV cannulae	14–24G and appropriate strapping
Equipment for monitoring airway, breathing and circulation	
Needles and syringes	1–50 ml
Sharps container	
Adult and paediatric intraosseous needles	or bone marrow needles
IV administration sets	including blood administration sets, high flow sets and buretrol
Stethoscope	
Pulse oximeter	with adult and paediatric probes
Non-invasive blood pressure monitoring device	including paediatric and large adult cuff sizes
Thermometer	including low reading capability
Blood glucose testing	
Collection tubes for investigations	

Appropriate hardware	
Heavy duty scissors to cut clothing	
Drip stand or equivalent hanging device	
Suction devices and suction catheters	rigid and flexible tips
Paediatric Broselow tape	
Fixation devices	adult and paediatric semi-rigid cervical collars
	head blocks
	spine boards
	restraining devices
	blankets and towel rolls
Universal precautions	gloves, goggles, gowns and face masks
Suture material	
Resuscitation trolley	capable of high Fowlers and Trendelenberg
Miscellaneous	
Resuscitation algorithms	
Resuscitation documentation record	

IV solutions
Ringers lactate or equivalent balanced salt solution
0.9% NaCl
5%, 10% and 50% dextrose
Paediatric solutions (e.g. half dextrose Darrows, Neonatalyte)

Essential drugs

If not specified, refers to parenteral formulation.

Category
Paralytics and sedatives
Suxamethonium
Pancuronium
Midazolam
Diazepam (PO, IV)
Etomidate
Ketamine
Anti-eptileptics
Phenobarbitone
Phenytoin (PO and IV)
Analgesia
Morphine (PO and IV)
Paracetamol (PO, PR and IV)
Diclofenac (PR and IV)
Lignocaine/Lidocaine
Cardiovascular
Adrenaline
Dopamine
Dobutamine
Atropine
Adenosine
Amiodarone
Diltiazem
Sodium nitroprusside
Nitroglycerine (PO, spray, IV)
Labetolol
Hydralazine
Frusemide (PO and IV)
Sodium bicarbonate
Vasopressin
Aspirin (PO)
Clopidogrel (PO)
Tranexamic acid
Vitamin K (PO and IV)
Potassium (PO and IV)
Antimicrobials
3rd generation cephalosporin (e.g. ceftriaxone)
Antipseudomonal antibiotic (PO and IV)
Clindamycin (PO and IV)
Doxycycline (PO)
Metronidazole (PO and IV)
Cotrimoxazole (PO and IV)
Chloramphenicol
Gentamicin
Ciprofloxacin (PO and IV)
Ofloxacin
Ophthalmic topical antibiotic
Antibiotic ear drops
Silverex cream topical
Fluconazole (PO and IV)
Artesunate (IV and PR)
Artemether
Quinine
Asthma
Ipratropium (Nebs)
Salbutamol (Nebs)
Hydrocortisone
Dexamethasone (PO and IV)
Prednisone (PO)
Magnesium sulphate
Electrolyte solutions
Calcium gluconate

Calcium chloride Sodium bicarbonate Magnesium
Anticoagulants Heparin unfractioned Enoxaprin Warfarin Streptokinase
GI drugs Ondansetron (PO and IV) Metoclopramide (PO and IV) Promethazine (PO and IV) Ranitidine (PO and IV) Activated charcoal Octreotide Insulin (soluble and Lente) Glucagon
Antidotes and vaccines Antivenom pentavalent for African snakes (as regionally appropriate) Pralidoxime Naloxone Protamine sulphate N-acetyl cysteine Neostigmine Rabies vaccine Rabies immunoglobulin Tetanus immunoglobulin
Other Thiamine Mannitol Diphenhydramine

346 Triage

Rudimentary emergency care systems tend to deal with patients in the order in which they present for care; as systems advance, formal medical prioritisation tools are used; very advanced systems in well-resourced settings now try to meet all demand by placing senior clinicians earlier in the care pathway.

Given that sick and injured patients present at any time, and that providers in our setting are often faced with more patients than they have resources to deal with, some form of prioritisation for care is required. Triage is that process, and refers to the systematic determination of the urgency of need for medical care; it rations patient treatment efficiently when resources are insufficient for all to be treated immediately. Remember that triage is dynamic: a patient's urgency for medical attention may vary with time. Always re-triage patients who are waiting to be seen.

The process involved varies contextually, depending on current demand and available capacity.

Event triage

During a major incident such as war or a tsunami, the demand for emergency care typically completely overwhelms capacity. The purpose of triage in such contexts is to rapidly identify the sickest in the crowd and ensure that treatment priority increases the likelihood of survival. This relationship is not always linear and triage here may include some patients that are too sick to justify treatment priority, as their likelihood of survival is poor.

Regardless of whether patients are being triaged in the field or at a facility, if a major incident or disaster has been declared, a rapid (and therefore less accurate) triage tool needs to be applied in order to quickly sort patients. Several tools exist but we recommend the Triage Sieve as primary triage (first pass) and the Triage Sort as secondary triage (more detailed assessment when more resources are available).

Facility based triage

Emergency visits are often unscheduled, undifferentiated and unpredictable in terms of arrival rates. This may initially be perceived as a mismatch of capacity and demand. However as standardised guidelines for prioritising

acutely ill patients on an individual basis are developed, more efficient use of resources follow. Facility based triage helps healthcare staff achieve this.

Triage is a process, not a place, but for the most part will need a dedicated area which allows for privacy. Triage nurses tend to be the first healthcare contact in most instances, although any grade of trained clinical staff can be used.

The South African Triage Scale (SATS) is an example of a triage tool for low resource settings, and has been shown to improve waiting times and patient flow. For online resources and further information visit: www.emssa.org.za/sats

Field triage

Most variability is found in this context depending on what resources are available. In some contexts the process may be very similar to event triage. Whilst this need is quite obvious during major incidents, it also has an important daily role in pre-hospital care.

The pre-hospital use of triage in the field varies from region to region. Such triage typically uses instability of vital signs to differentiate high from low priority patients. Discrepancies in triage appear when personnel of differing levels of medical experience and qualifications need to assess patients, as there are no clear definitions of 'unstable' physiology. The terms 'stable' and 'unstable' are poorly understood and fail to accurately reflect the patient's clinical condition.

Several tools exist, none of which have all of the ideal characteristics for triage in this environment. For trauma, tools such as the Triage Revised Trauma Score may be helpful but they have limited applicability in children and none in medical patients. Some settings use a modification of the SATS, but it has yet to be validated for this environment.

Accurate pre-hospital triage is essential for appropriate call out of secondary resources, accurate notification of receiving hospitals, and quality assessment and audit of the ambulance services. Triage tools based on objective discriminators are essential.

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Part

7

B. Out of hospital care

347 General principles of pre-hospital care

Pre-hospital care covers a broad spectrum from basic first aid (e.g. bandaging a wound) to advanced clinical interventions (intubation or thrombolysis). The scope is largely determined by local training and clinical practices.

Two commonly cited models are Franco-German and Anglo-American – depending on whether the emergency medical service (EMS) is a physician led or EMS practitioner led. We discuss the underlying principles that drive the establishment of an EMS.

Emergency medical services system construction

A well-constructed EMS system will ensure that patients reach appropriate care in a timely manner. The capacity of a society to afford expensive technologies should not preclude the adoption of systems principles. The essential components of an EMS system are the following:

- Telecommunication access to a contact centre and a command and control structure
- Community response
- EMS response
- Medical rescue services
- Patient transport services

Access

Patient access should preferably be via an easy three digit universal emergency number, such as 112 or 911. Most countries have cellular networks and most emergency requests will come via cellular devices. For rationalisation, triage and coordination of response, emergency calls should be received and dispatched by regionalised centres.

Community response

The community capacity to provide emergency care must be developed to provide for recognition of medical emergencies, effective relay to formal EMS, and early basic first aid.

EMS response

The core of pre-hospital emergency care is the ambulance, which can be in any appropriate format e.g. 4 × 4 vehicle, bicycle trailer. Ambulance units consist of vehicle, trained personnel, equipment, communication devices and medication.

Medical rescue services

Medical rescue services establish access to patients, release them from entrapment and provide medical care in the

process. Traffic accidents are the commonest cause of patient entrapment, but medical rescue may be needed in other environments (e.g. wilderness) where conditions threaten health.

Patient transport services

Without dedicated patient transport systems for non-emergency patients ambulances can be consumed doing non-urgent work and be unavailable in emergencies.

Principles of pre-hospital care

Definitive care

Patient outcome depends on access to definitive care. Pre-hospital care must therefore focus on the transfer of the patient to definitive care; field stabilisation is senseless if the patient lies at the side of the road never to reach a hospital.

Time

Patient outcomes depend on the time of access to emergency services, admission to an emergency facility, and admission to definitive care. Pre-hospital systems often focus on overall response times, but each element of response has impact:

- Time of access to a contact centre
- Time of dispatch of ambulance
- Time of arrival of scene
- Time on scene
- Time to hospital
- Time at hospital
- Time to definitive hospital care (inter-hospital or inter-department transfer)

Decreasing time consumed in each phase improves the chances of a unit being available for the next response.

Minimum standards

Pre-hospital systems must set standards against which to measure capacity and performance. Standards should exist for:

- Personnel
- Equipment
- Medication
- Vehicles
- Performance

Operating procedures

Services should have standard protocols and procedures that guide practice.

Medical oversight and quality management

Clinical oversight is critical when designing EMS systems. This may take the form of either emergency physicians with pre-hospital experience or highly qualified, experienced pre-hospital practitioners. Regardless of which model is used, the establishment of multi-disciplinary clinical governance structures should be encouraged. This permits quality of care to be examined throughout the care pathway.

Command and control

A system requires strong leadership for operational and clinical decision making. A geographic model of control is a good one to follow, meaning that an individual controls people, processes and technology of a service within a

defined manageable geographic area.

Staffing of ambulances

The crew of an ambulance is often determined not by actual staffing needs but by who is available. EMS is the link between a patient and a health facility. This implies two aspects: transport and care, and a minimum of two people are needed – a licensed driver and somebody to look after the patient.

In the absence of formally trained ambulance personnel, consideration should be given to utilising trained community volunteers. Organisations such as the Red Cross and Red Crescent as well as other non-governmental agencies can assist with first aid training for such volunteers.

Remarkably, while these are fairly minimum requirements for an ambulance service, experience shows that 80% of patients will be safely conveyed to a health facility, regardless of level of service provided.

348 Transfer medicine

Definition and principles

Inter-facility transfer is often required for definitive care and may be executed by land, air or sea. Transfer medicine is a young speciality; its aim is to prepare clinical personnel to assess and stabilise patients for transport and address emergency conditions that occur in route.

The 3 Ws of transfer medicine

Where should I transfer the patient?

'Blind referrals' are bad referrals; clinical details should be discussed with an accepting provider at the receiving facility to ensure that higher-level resources are available, and a transport plan developed. The receiving hospital must be both willing and able to treat the patient. Even if a hospital must decline a referral, they should be able help identify an alternative.

Which information should I give the receiving doctor?

- Patient's name and date of birth
- Brief presenting history, list of ALL diagnoses
- Vital signs, O_2 sat and GCS
- Current therapies, including drugs, fluids, and whether patient is O_2-dependent
- Procedures performed and procedures needed
- **Always review reason** for transfer (to ensure that the needed services are actually available at the receiving facility)

What must be done before transfer?

- ABCs performed and reviewed just prior to departure
- Prepare a summary of clinical course, including any diagnostic studies done (labs, pregnancy test, ECG) with results (clearly note any pending results and include a phone number receiving provider can use to follow up)
- Wound dressings, splinting where indicated (rides can be very bumpy)
- Check that all lines are secure
- Ensure that patient is adequately monitored en route

Never delay transfer for diagnostic results.

349 Aero-medical services

Aero-medical services bring providers to patients and patients to definitive care, and can dramatically shorten time to life saving interventions. However, they are expensive and are not affordable in all systems. Health care systems

should have aero-medical utilisation guidelines to ensure clinical benefit by:
• Shortening time to definitive care
• Providing specialised medical expertise or equipment
• Providing transport to otherwise inaccessible patients

The decision to activate aero-medical resources is medical, and should be made separately from the determination of whether the flight can be undertaken safely.

The scope of aeromedical response

• Scene response: aircraft can travel to incidents quickly (providing there is a place to land) allowing skills and equipment to be rapidly deployed
• Inter-facility transfer or repatriation: aircraft can be used for rapid transport between facilities
• Technical rescue: specially equipped helicopters can be used to rescue patients from mountains, water, ships and buildings; if they are fitted with a hoist and sling and are crewed by trained personnel, rescue is possible even where the aircraft cannot land
• Clinical outreach services: aircraft are also used to transport health professionals to render services in remote areas

When to fly

In remote areas, air transport may be the only practical option to transport a patient to definitive care within a reasonable timeframe and rigid criteria may not be relevant. Common indications include:
• Medical criteria:
  » Head injuries with GCS 6–12
  » Spinal injuries
  » Respiratory distress despite full O_2 therapy
  » Severe penetrating injury
  » Obstetric and gynaecological emergencies
  » Neonatal emergencies
• Logistical criteria:
  » Extrications that would otherwise exceed 45 minutes
  » Limited EMS access to patients
    • Major incidents and mass gatherings
    • Outreach programmes.
• Patients with special conditions

Packaging the patient for flight

There is limited space and access to a patient in an aircraft, so adequate stabilisation prior to flight is critical (sedate anxious patients; intubate, etc.). The aim is to predict what may go wrong during flight, and prevent it. Monitoring is almost entirely electronic as noise and vibration substantially limit physical exam.

Choosing the appropriate aircraft

Helicopters and fixed wing aircraft provide services.

Advantages of helicopters	**Disadvantages of helicopters**
• Remote area access • Ability to hover for rescue • Can land in most areas	• Small working space • Effects of altitude • Range limits • Vibration and noise
Advantages of fixed wing	**Disadvantages of fixed wing**
• Speed and long distance	• Landing strip needed

• Larger cabin work space • Cabin pressurisation • Less vibration than rotary	• Effects of attitude (if unpressurised)

Aircraft operational range

Aircraft are limited by the fuel load, speed and the amount of weight they can carry. This will dictate the mission range. In general terms, range before refuelling:
• Helicopters: 250 km
• Unpressurised light aircraft: 250 to 400 km
• Pressurised aircraft: > 400 km
• Medium jets: > 1 500 km

350 Basic emergency first responder kit

Determining the essential content of a basic emergency kit is affected by many factors, including setting, provider skill level, and local burden of disease. This basic list gives responders a guide to core equipment that should be readily available during an emergency field response. Ensure that there is a suitable way of storing and carrying the equipment: waterproof nylon satchel or backpacks are commonly used. Ensure that equipment is checked and cleaned on a regular schedule.

Basic emergency care kit for a first responder

General

• Trauma scissors
• Forceps
• Thermometer (low reading)
• Tourniquet to facilitate IV placement
• Stethoscope
• Blood pressure cuff
• Latex gloves
• Eye protection
• Torch
• Water

Circulation management

• Ringer's lactate
• Normal saline
• Blood administration sets
• Fluid administration sets (15, 60 drops/ml)
• IV cannulas (24–14G)
• 'Esmarch' or similar rubber bandage tourniquet
• 75 mm bandages
• Wound dressing pads 100 mm × 100 mm
• Wound dressing pads 100 mm × 200 mm
• 75 mm Elastoplast adhesive bandage
• Alcohol antiseptic swabs
• Triangular bandages or linen strips

Airway management

• Oropharyngeal airways full size range

- Pocket mask
- Hand-held suction device

Breathing management

- Bag valve mask (infant, child, adult)
- Non-rebreather oxygen mask
- Nebuliser mask
- Supplemental O_2 source

Disability management

- Padded wire splints
- Cervical collar (adjustable)

Special conditions

- Space blanket
- Hydrocolloid burn dressings
- Glucose for oral administration
- Eye patch

Part

7

C. Disaster medicine

351 General principles of disaster medicine

The word disaster means different things to different people, and the impact of similar events differs greatly between countries. There has been an increase in the rate of disasters, including a four-fold increase in weather-related disasters. Global urbanisation is an important factor.

Definition

WHO defines a disaster as 'an event of a magnitude resulting in a level of suffering which overwhelms the local community response'.

A major incident is defined as one where the number, type or severity of live casualties, or location, requires special response measures. Major incidents may be simple (infrastructure (roads, bridges etc.) remains intact) or compound; compensated (enough medical resources can be brought in to deal with the response) or uncompensated. An uncompensated major incident is a disaster.

Types of disasters

Natural or man made

- Earthquake
- Floods
- Droughts
- Fire
- Tornados

- Transport accidents
- CBRN chemical, biological, radiological and nuclear
- Structural collapse
- Civil unrest and conflict

Phases of a disaster

Managing disasters requires an understanding of the role of the medical community in each phase.

Pre-disaster

Prevention, mitigation, preparedness and early warning. The ability and efficiency of the health response is dependent on adequate planning. Hospital plans and simulation exercises are essential.

Post-disaster

Response, recovery, rehabilitation, reconstruction. Search and rescue of casualties, transportation to hospitals and treatment are the main activities for the health community. Recovery and rehabilitation of staff, hospitals and health systems is critical in ensuring on-going community service in this period.

Disaster medicine

Disaster medicine includes not only responding but also ensuring preparedness for disaster response.

- An integrated response – planning and responding require a multi-disciplinary approach. Health needs must be integrated into the plans of other response agencies.
- All hazard approach – operational planning and response is based on capability rather than the particular hazard.
- Incident command system – an ICS is the cornerstone of managing such events. A predefined command structure must be in place and agreed upon by all the relevant parties.
- Triage – in a disaster the aim shifts from doing the most for every single patient to doing the most for the most. To achieve this, a triage system must be used to ensure that limited resources are used appropriately.
- Communication often fails in disasters. Robust plans should be in place to ensure both internal or institutional, and inter-agency communication.
- The plan – government, hospital and EMS plans all need to be living documents. Plans need the input of all relevant parties so they will be aware of their specific responsibilities when the plan needs to be activated.
- Review of plans – to ensure that plans remain relevant and current. This could be done annually or at another agreed upon interval. It is important that the plan is reviewed after each incident.
- Training is important to ensure currency of the staff expected to respond to a mass casualty incident. Training should include some sort of exercise. These can take the form of large-scale simulation exercises, which are often resource intensive. Another option is table top exercises.

Helpful mnemonics

Priorities for response can be recalled by CSCATTT:

- Command and control
- Safety
- Communication
- Assessment
- Triage
- Treatment
- Transport

Communication is usually the biggest failing in disaster response. METHANE helps ensure appropriate communications:

- My call sign
- Exact location
- Type of incident
- Hazards on scene
- Access (and egress routes)
- Number of casualties
- Emergency services (on scene and needed)

352 Complex humanitarian emergencies

Complex humanitarian emergencies involve a significant disruption of social services resulting in medical and public health emergencies, with insufficient resources to deal with the consequences. They are typically due to a significant breakdown in governmental authority, as a result of internal or external conflict.

Citizens often experience collateral damage due to:

- Indiscriminate targeting by forces
- Voluntary or forced displacement to avoid conflict
- Severe disruption of social services, including health care
- Targeting of health care systems

A number of international organisations such as WHO, ICRC and MSF have developed specific strategic responses to these types of emergency.

Legal protection

International Humanitarian Law, including the Geneva Conventions and associated protocols, make the following provisions for the ill and injured:

- They must be provided with medical care and attention, to the extent possible, with the least possible delay and without any distinction on non-medical grounds
- They must be sought and evacuated (to the extent possible)
- They and health-care personnel carrying out their exclusively humanitarian task must not be attacked or ill-treated
- The passage of medical transports must not be restricted
- Health-care personnel must not be punished for carrying out activities compatible with health-care ethics – they must be assisted

Even though most states are signatories to the Geneva Conventions, the above prescripts are violated and health workers, including international responders, may themselves become targets.

Ethical considerations

Impartiality, confidentiality and patient rights remain cornerstones in treating any patient. Health care providers may often find themselves in a dilemma when treating patients seen to belong to an opposing faction in the conflict.

War zones often do not distinguish between combatants and civilians, and health care workers are likely to have to treat both groups. Keeping patient confidentiality may become very difficult, and there may even be laws in place compelling a health worker to divulge information to the authorities.

A health care provider must be mindful of the following:

- Patient confidentiality must be maintained at all times
- A patient should receive the best treatment possible with the resources available
- Patients and families have the right to be treated with dignity and respect
- Medical ethics should be considered at all times
- Patients, irrespective of colour, creed or allegiances must be treated impartially

In a conflict zone, providers may find themselves caught in cross fire, treating combatants or civilians from an opposing forces, being called upon to disclose patient information or allow access to opposing force patients, and not having sufficient resources or services to adequately provide the required treatment. In such circumstances a provider can only do the best with what is available, and may have to withdraw from a situation and even abandon patients due to extreme danger. Provider safety and survival is essential in order to ensure health care viability.

353 Pre-hospital disaster response

Pre-hospital disaster management is extremely difficult with limited resources or in remote environments. The concepts below provide an easily remembered, generic approach.

Generic concept

The ‘**FACE**’ diagram is easily remembered by responders and easily plasticised for use as an on-scene aide. Principles of the plan are outlined and listed in numbered sequence to help responders create order out of chaos: the central starred incident (‘nose’) is the first to occur (e.g. explosion). First and subsequent medical responders need to

create.
• **FCP (forward control post)**: (mouth) provides overall on-scene command and control, safely positioned, incorporating all senior medical and essential responding agencies (e.g. police, fire). The FCP is the hub for all communication to other agencies, hospitals and the media.
• **IC (inner cordon) and OC (outer cordon)**: ('face') these are two security cordons set up around the scene to protect victims and rescuers, and provide an adequate space for responders to work.
• **FAP (first aid post)**: ('left eye') – in most disasters, 80% of the patients on scene are able to move by themselves (**green** triage) – these patients can be directed and assisted to the FAP.
• **CCS (casualty clearing station)**: ('right eye') 20% of patients left within the inner cordon are either dead or seriously injured. These patients are moved to the CCS, where the most on-site medical expertise/equipment is concentrated to provide the best care with the resources available. After being stabilised, patients can be transported to receiving hospitals. The dead remain at the incident site, where they were found.
• **CHA (central holding area)**: ('neck') is an area for the receiving, sorting and dispensing of additional resources (medical equipment, ambulances or additional medical staff).
• **Access ('TO') route**: must be secured very early by police or military authorities to prevent it from being blocked by crowds of onlookers. Between the access and egress points, loading of patients may begin from a point near the CHA.
• **Egress ('FROM') route**: this route to move patients away from the incident site is as important as the access route. Authorities need to secure this route early to ensure that bnefits of carefully prepared evacuation are not negated by a chaotic blockade.
• **Communications**: ('earphones') adequate communication on the scene and to outside agencies is critical. Cell phones are used frequently in Africa; however, networks are prone to become overloaded in disasters. The expectation should be that cell phones will not work during a disaster. Many other communication options exist, such as two way radios, runners or satellite systems. Whatever system is used, it needs to be tested regularly.
• **Hospitals**: these are either the **primary** (nearest hospital) or **secondary** (backup for overflow patients). The aim though is to refer patients *appropriately*, e.g. vascular injuries may have to bypass local facilities, for a more distant specialist hospital. Essential communications from the FCP to the Ambulance Control should be passed on to the receiving hospitals ahead of time, so they may anticipate and plan for the injuries and numbers of patients arriving.

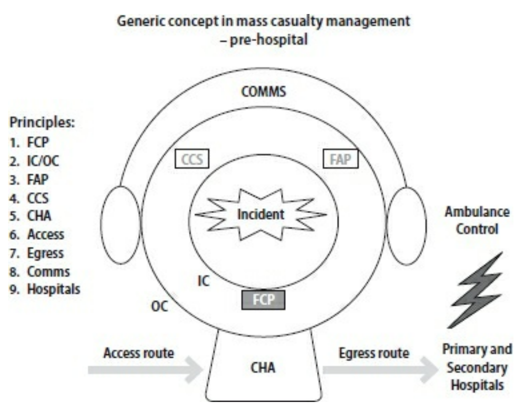

Figure 353.1: Schematic outline of a generic pre-hospital disaster plan

354 Hospital disaster response

Disasters result in large numbers of patients arriving simultaneously at a hospital, disrupting functioning at a moment's notice; planning beforehand is essential to ensure optimal functioning, and consists of four steps:
• **Assessment** of needs and estimating the number of patients that need to and can be managed by the facility
• **Planning** to ensure optimal facility function under abnormal circumstances
• **Implementing** the plan in the event of a disaster
• **Evaluating** to ensure readiness continually

Determining flow of patients and identifying areas

A hospital has to rapidly expand and reorganise its capabilities to meet sudden demands. Flow from entrance to disposal must be logical and sequential.

Required predesignated areas required (see Figure 354.1)

- Ambulance and vehicle patient drop-off point
- Reception and triage area
- Priority 1 resuscitation area
- Priority 2 treatment area
- Priority 3 waiting and treatment area
- Body holding area (mortuary area)
- Admission area(s)
- Information point and media briefing area
- Discharge and reuniting area
- Staff rest area

Surge capacity

Determine what additional local space can be utilised (parking garages, recreation halls, etc.) and how many patients can be accommodated.

Action cards

Compile an action card for each staff member specifying the actions to be executed. Include a map/plan of the area on the card.

Determining equipment and resources required

Plan the equipment, medicine and consumables required for the number of patients in each area.

Activating the plan

- The telephone exchange, emergency personnel and the after-hours senior nurse should be trained to activate the plan
- Record information from the caller/informant as well as all available information on the event
- Communicate this immediately to a predetermined decision maker

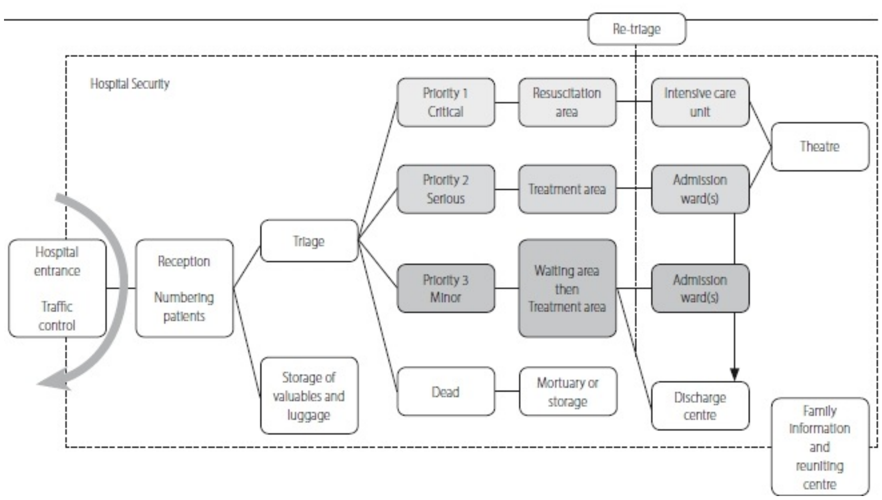

Figure 354.1 Flow of disaster patients within hospital

Based on the information available the decision maker must decide to:

- *Put the hospital on stand-by:* the hospital may receive an influx of patients. Normal routines continue but a rapid assessment is done of available capabilities
- *Activate the disaster plan:* All areas are activated and command staff are called to their stations. All staff

members report to their pre-allocated positions; unallocated staff report to a central allocation point

Five-minute disaster plan for a rural hospital

- Decide who is in command
- Close the gates and entrances; channel all patients to a single entrance
- Call additional staff
- Allocate current staff for:
  - Triage
  - Priority 1 treatment
  - Priority 2 treatment
  - Priority 3 treatment
  - Wardcare
- Number all patients on arrival with an identification band
- Triage patients and mark with colour-coded triage tag
- Channel patients to applicable treatment area
- Accept only Priority 1 patients in the resus room, and resuscitate
- Identify area for Priority 2 patients, and treat
- Channel Priority 3 patients to a waiting area and appoint staff to continuously monitor their vital signs – treat after Priority 1 and 2 patients
- Request only diagnostically significant investigations
- Ensure proper recordkeeping; mark all documents, valuables and records with patient's disaster number
- Prepare ward for admissions and channel all stabilised patients to ward(s)
- Apply surgical triage and compile theatre list
- Start surgery as soon as feasible and ensure post-op care
- Compile a list of all patients' names against their disaster numbers
- Place staff members at gates to manage enquiries about patients
- Assemble all bodies in a practical area for management by police
- Ensure re-assessment of discharged patients
- Transfer patients with a poor prognosis to a higher level of care

Evaluating the plan, and preparing for the next event

The current plan's strengths and weakness should be evaluated after each event and the plan revised as needed.

To ensure that staff are prepared and that challenges are identified in a timely manner, communication exercises, and table top and full simulations need to be conducted at regular intervals.

355 Ethics in disaster medicine

Disasters present clinicians with many ethical dilemmas due to an acute imbalance between medical needs and medical services available. The overarching principle remains for the practitioner to assist in the relief of human suffering and alleviation of pain.

Ethical considerations

Duty to care

The American Medical Association states that '*individual physicians have an obligation to provide urgent medical care during disasters*'. This holds even in the face of greater-than-usual risk to their safety, health or life.

Triage

In disasters the emphasis moves from maximising benefit for every individual to attempting to do the most for the

most. This may conflict with the principle of non-maleficence, as not all victims will be treated immediately. However, the principle of justice requires that resources must be distributed equally. Triage is the tool to achieve this, and it must be used without any consideration to race, age, gender or creed. It is unethical to use limited resources to save one patient when such resources could have been deployed to save many more victims. Ethical principles in fact do not change, but there is a shift from individual ethics to collective ethics.

Informed consent

In a disaster it may not always be possible to obtain informed consent, and implied consent is often presumed.

Media

The media rapidly descend on disaster sites and can be friend and foe. If well managed they can keep the community informed and relay key instructions. Cameras may pose a serious risk to privacy and confidentiality. Medical personnel must protect victims at all times.

Sustainability

Responsible disaster management includes attention to sustainability. If all teams withdraw after the acute phase with an inadequate hand over plan (as in the 2010 Haiti earthquake), the benefits of the initial response may be lost.

356 Chemical and biological incidents

Chemical

There are three key components to the management of patients affected by chemicals.

Identify contamination

Patients are generally contaminated with the chemical(s) that affected them and pose a threat to providers.

- Undress the patient before moving into a confined space – undressing removes about 70% of contamination
- Brush off powders; dispose of clothes appropriately
- When feasible, rinse the patient with high flow water before starting treatment

Identify the substance

In most cases the causal substance is not known. EMS or fire personnel may know. Assume high toxicity if unknown.

Chemical effects and injuries

Life threatening conditions (due to chemicals or other injuries) must be managed first.

There are no specific antidotes for most chemicals, and exposure should be treated symptomatically. See 📖 Approach to acutely poisoned patient, p. 652. See 📖 Cyanide poisoning and organophosphate poisoning, p. 700 and p. 714.

Biological

Biological incidents include naturally-occurring outbreaks of infectious disease as well as incidents of an infectious disease, or caused by intentional dissemination of a biological agent (e.g. the spread of anthrax spores).

Disease outbreak

The majority of malicious acts with biological agents will present and are managed in the same manner as naturally occurring outbreaks. In most cases the fact that the outbreak is not the result of a natural occurrence will only be realised at a late stage (see 📖 Outbreak response, p. 922).

Decontamination

The possibility of incidents caused by known dissemination of biological agents that require decontamination processes is very low (e.g. the letters contaminated with anthrax spores in the USA during 2001).

Important considerations:

- The possibility of the incident being a hoax is very high; have a sample of the powder or other substance analysed at a laboratory as soon as possible
- The emergency services who manage the scene should take samples and have them delivered to the correct laboratory
- All persons who have been exposed to the substance must be identified and observed for two weeks or until analysis results are negative
- In cases where anthrax has potentially been used, everybody who has been in direct contact with the powder must be washed with soap and water and have their clothes washed
  - » They should be put on prophylactic amoxicillin or doxycycline for two weeks or until the analysis results are negative

357 Mechanical and structural collapse

Recent rapid urbanisation, especially within low income countries, brings many risks. When there is poor implementation and enforcement of building codes, mechanical or structural failure may occur, with the potential for a large number of casualties.

The primary aim of the structural collapse response is to rescue survivors and offer definitive care as soon as possible. Responders will be faced with a difficult environment, including conditions of up to complete darkness. Access to patients can be poor and working space very limited. Collapse response requires well-prepared medical responders who remain vigilant in the hazardous environment.

Types of structural failure

Two broad categories exist:

- Foreseeable collapse of one or two structures as in the case of old or damaged buildings
- Sudden and simultaneous damage to and collapse of multiple structures, at more or less the same time results in a large number of casualties, and often secondary to some other large event, such as an earthquake

Impact of structural collapse

Buildings will collapse in different ways depending on which structural elements fail. Public utilities such as transport, electricity, and gas lines may be affected. The most important consideration at the collapse site is the possibility that persons are trapped.

Scene safety

The safety of responders is critical. If you feel unsafe then you probably are. Don’t enter areas where you may become a victim. Personal protective equipment is mandatory. Scene hazards are numerous and include:

- Toxic fumes
- Fire
- Hazmat conditions
- Secondary collapse
- Electric cables and overhead power lines
- Falling structures
- Disrupted gas or liquid fuel lines

Command and control

The response to a structural collapse will most likely include many emergency agencies, and needs a pre-determined Incident Command System (ICS). The responsibility of an ICS is overall control of the incident. CSCATTT principles apply (see 🕮 Pre-hospital disaster response, p. 906).

Possible location of victims

Depending of the type of structure, survivors may be trapped in voids – small spaces created by the collapse. Common areas in a building where voids are created include:
- Basements and cellars
- Elevator shafts
- Bathrooms
- Next to concrete walls that have remained standing
- Next to chimneys or fireplace
- Rooms that survived destruction

Injury profile

The nature and extent of possible injuries depends on a number of factors, including the structure that collapsed, its construction material and the length of time that the victims are entrapped. Common injuries include:
- Airway contamination (smoke, dust or other particles)
- Crush syndrome – rhabdomyolysis, hyperkalaemia and renal failure
- Hypothermia or hyperthermia
- Long bone fractures
- Partial or complete amputations
- Soft tissue injuries cause by compressive forces
- Dislocations and lacerations

358 Mass gatherings

Typically considered to be an event with over 1 000 in attendance, mass gatherings have an increased risk of illness and injury compared to the general population.

Risk assessment

The number and type of medical support needed at mass gatherings has historically been based on the number of attendees. Recent studies show that a multi-factor score best predicts risk and needed personnel.

Determination of required medical resources based on the risk

Table 358.1 shows a model for estimating the medical resources that should be placed at mass gatherings in Africa, although clearly the resources deployable will vary from country to country.

Within each category, risk factors are allocated a score. Only one score is considered per category (e.g. a New Year's celebration may also include a pyrotechnic display; the category will be scored 7, as it is higher than the 4 allocated to pyrotechnics).

Category L identifies those factors that may lower patient presentation rates.

Table 358.1: The medical resources needed at a mass gathering in Africa

(A) Nature of event	Classical performance	2
	Public exhibition	3
	Pop/rock concert	5
	Dance event (rave/disco)	8
	Agricultural/country show	2
	Marine	3
	Motorcycle display	3
	Aviation	3
	Motor sport	4
	State occasions	2
	VIP visits/summit	3
	Music festival	3

	International event	3
	Bonfire/pyrotechnic display	4
	New Year celebrations	7
	Demonstrations/marches	5
	Sport event with low risk of disorder	2
	Sport event with medium risk of disorder	5
	Sport event with high risk of disorder	7
	Opposing factions involved	9
(B) Nature of venue	Indoor	1
	Stadium	2
	Outdoor in confined location, e.g. park.	2
	Other outdoor, e.g. festival	3
	Widespread public location in streets	4
	Temporary outdoor structures	4
	Includes overnight camping	5
(C) Seated or unseated	Seated	1
	Mixed	2
	Standing	3
(D) Spectator profile	Full mix, in family groups	2
	Full mix, not in family groups	3
	Predominately young adults	3
	Predominately children and teenagers	4
	Predominately elderly	4
(E) Past history	Good data, low casualty rate previously (less than 0.05%)	–1
	Good data, medium casualty rate previously (0.05% – 0.2%)	1
	Good data, high casualty rate previously (more than 0.2%)	2
	First event, no data	2
(F) Expected numbers	< 1 000	1
	1 000–3 000	2
	3 000–5 000	4
	5 000–10 000	8
	10 000–20 000	16
	20 000–30 000	20
	30 000–40 000	24
	40 000–50 000	28
	50 000–60 000	32
	60 000–70 000	36
	70 000–80 000	42
	80 000–90 000	46
	90 000–100 000	50
	100 000–200 000	60
	200 000–300 000	70
(G) Expected event duration (including queuing from gate open time)	Less than 4 hours	1
	More than 4 less than 12 hours	2
	More than 12 hours	3
(H) Season (outdoor events)	Summer	2
	Autumn	1
	Winter	1
	Spring	1
(I) Proximity to hospitals	Less than 30 min by road	0

(nearest suitable emergency facility)	More than 30 min by road	2
(J) Profile of hospitals	Multiple acute care areas	1
	Large acute care area	2
	Small acute care area	3
(K) Additional hazards	Carnival	1
	Helicopters	1
	Parachute display	1
	Street theatre	1
	Water hazard	1
	Onsite alcohol use	1
(L) Additional on-site facilities	Suturing and or plastering	2
	Vending machine for over the counter medication	2
	Public access AED	1
	Existing full time operational medical facilities on-site	2

Calculation of the event’s risk score

Risk score = Sum of scores (categories A–K) – score (category L)

The resultant score is then referenced against the resource matrix as defined in Table 358.2.

Table 358.2: Resource matrix

Score	Ambu-lance	BLS	ILS	ALS	Ambu-lance crew	Doctor	Nurse	Coordi-nator
<20	0	2			0	0	0	0
21–25	0	4			0	0	0	0
26–30	1	4	1	0	2	0	0	0
31–35	1	6	1	1	2	0	0	visit
36–40	1	8	1	1	2	0	0	visit
41–45	2	12	1	1	4	1	0	1
46–50	2	16	2	2	4	1	1	1
51–55	3	20	3	3	6	2	1	1
56–60	3	24	3	3	6	2	2	1
61–65	4	32	4	4	8	2	2	1
66–70	5	40	5	5	10	3	3	1
71–75	6	48	6	6	12	3	3	1
76–80	8	64	8	8	16	4	4	1
81–85	10	80	10	10	20	5	5	2
86+	15	120	15	15	30	6	6	2

Example : An event scoring 47 will need the following medical resources on site:

- 2 ambulances
- 16 Basic Life Support/First Aiders
- 2 Intermediate Life Support
- 2 Advanced Life Support
- 1 Doctor
- 1 Nurse
- 1 medical coordinator on site

359 Recovering from a disaster

Recovery is the process of returning individuals or communities to their pre-disaster state. It is the least researched aspect of the disaster cycle (preparedness, response, mitigation, recovery). A pre-event plan detailing priorities for reconstruction will prevent delays and ensure that vulnerabilities in the system are dealt with.

Recovery as a process

Recovery is not a singular event, but multiple parallel processes often involving different time frames. Components include infrastructure (roads, sewerage, water, electricity etc.), economic centres (including lost equipment, stock, livestock and feeds), housing, psychological recovery and a re-evaluation of disaster planning and response. In complex humanitarian disasters involving refugees and internally displaced people, the boundary between having an on-going disaster and a protracted recovery period is blurred, with generations of young people born away from their country or city.

Four overlapping phases are recognised: the initial emergency, restoration, replacement and development. The way in which these phases are tackled is dependent on the competing interests of wanting to (i) rapidly return to normal, (ii) use the destruction as an opportunity to increase safety and (iii) improve the community. Usually, rapid return to normality is the aim.

Sustainable development

The incorporation of sustainable development in post-event recovery is only possible with pre-event planning; recovery from an event should leave a community stronger and more resilient than it was before. It requires a hazard and risk assessment, identification of alternative building sites, and codes and zone laws which could be brought into play in the recovery.

A sustainable development plan may involve shifting residential or commercial areas and/or requiring a higher standard code involving better or more expensive materials in the reconstruction of buildings; such a process may take longer and initially be more costly. However, it should reduce effects and costs in the long run. In many instances, existing structures, such as buildings, dams, dykes or bridges are only built with a specific life span or for a given level of disaster severity (e.g. a level 7 earthquake).

Recovery of the local health system

How the local health system will cope depends on many factors, such as type of event, structural involvement of treating facilities, effect on healthcare personnel and the damage to supply lines. Short-lived, well demarcated events (such as a building collapse or explosion) may temporarily overwhelm local resources but the number of casualties is limited. The nature of the event usually means at least a selection of health care facilities and infrastructure are in place, even though healthcare staff may be working extended and rapidly cycling shifts. During a protracted event, assuming that personnel are not affected and absenteeism is not a big issue, the local personnel response will usually deteriorate after 72 hours. In a large scale event (such as a tsunami), a number of facilities may also be taken out of commission.

Similar to the preparedness for disaster response, plans should be in place for pre-event credentialing of volunteers, medical practitioners and nurses, so that they can be called upon in the recovery phase. More than dealing with disaster-related injuries, the largest burden of disease will now be from neglected chronic medical conditions and new day-to-day injuries and conditions.

360 Outbreak response

Hospital emergency areas are often where the first cases in an outbreak of a communicable disease will be seen, yet initial cases often go unrecognised or are misdiagnosed (missing an opportunity for rapid response and control measures). Often the specific aetiology may not be identified, but an unusual occurrence of certain syndromes alerts health professionals to a possible outbreak.

Standard infection control practices will protect against many agents. Outbreaks as a result of intentional release of agents are particularly difficult to recognise early as presenting features may be non-specific or the agents used unusual (and the resulting clinical syndrome is not recognised).

What is an outbreak?

- Two or more people with the same disease or symptoms or organism linked through common exposure, personal characteristics, time or location
- A greater than expected rate of infection compared with the usual background rate for the particular place and

time among a specific group of people
• A single case of a rare disease with epidemic potential in an area where this disease is not endemic, e.g. cholera
• A single case of a serious disease with potential to spread (particularly in health settings) e.g. a viral haemorrhagic fever

'Pseudo-outbreaks' can lead to misguided interventions and control measures. They may result from:
• Changes in reporting patterns, changes in size of at-risk population
• Media 'outbreaks'
• Laboratory issues: incorrect choice of test, incorrect interpretation of results, quality issues, testing errors, etc.

Steps in outbreak investigation

• Establish that a problem exists – confirm outbreak
• Confirm using appropriate lab tests where possible
• Alert public health authority rapidly
• The following steps will probably be carried out by the 'outbreak team':
  » Define a case (case definition may be broad, includes key signs and symptoms, time and place and epidemiologic links), active case finding, collection of data, descriptive epidemiology, develop hypothesis

Common syndromes associated with outbreaks

Table 360.1: Common syndromes associated with outbreaks

Syndrome	Differential diagnosis	Sample and investigations	Prevention and control measures in hospital	Comments
Acute onset painless watery diarrhoea in adult/s with dehydration	Consider cholera (1 case = outbreak)	Fresh stool for processing within 2 hrs, or use transport medium (e.g. Cary-Blair) If stool unobtainable rectal swab Microscopy culture and sensitivity (MCS), including for cholera	Standard and contact precautions when managing patients	Report urgently to local authority – need to establish likely source and prevent further cases due to contaminated water (usual source)
> 2 cases of diarrhoea with blood linked by place, person and time	Dysentery *Shigella*, *Salmonella* (including typhoid), *E. coli* (including EHEC)	Fresh stool for processing within 2 hrs; or use transport medium (e.g. Cary-Blair) MCS	Standard and contact precautions	
Acute onset fever, intense headache ± stiff neck ± rash	Meningococcal disease Rapid progression typical within 24 hours	CSF and/or Blood for MC&S Gram stain of CSF MCS Latex agglutination	Standard and droplet precautions	Report urgently to local authority Prophylaxis with ciprofloxacin – stat dose (or ceftriaxone) to close household contacts, crèche, medical staff exposed to respiratory droplets
Acute onset fever and bleeding (< 21 days)	Viral haemorrhagic fever (VHF) accurate history (within previous 21 days, probable exposure to ticks, animal tissue, rodents, bush meat, bats; travel/residence in endemic region in a country; occupational risks – health care worker, animal worker – vet, abattoir, farmer) Broad differential	Screening bloods, WBC platelets. LFTs – AST/ALT Specific VHF test and tests for alternative diagnosis	Strict barrier nursing Report urgently to local authority	
Clusters of patients or health workers with acute onset fever headache, myalgia respiratory symptoms, ±diarrhoea	Differential broad influenza (including novel subtypes) Severe Acute Respiratory Infection; new and emerging respiratory agents	Naso-pharyngeal/ oropharyngeal swabs placed in viral transport medium – alert laboratory	Standard, droplet and aerosol precautions	

Note: see specific Infectious Disease chapters for treatment, pp. 310–390

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Part

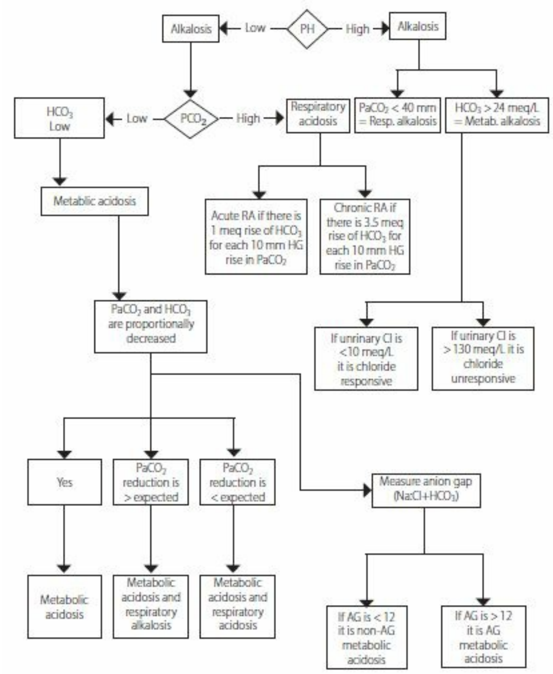
8

Appendix

Acid-base algorithm

Normal blood gas values:

pH 7.35–7.45
HCO_3 15–24 meq/L



Age-based vital signs

In an emergency, the following formulae may help in children aged 1–10:

Systolic BP = 90 + (2*age)

Weight = 2*(age+4)

Age	Wt (kg)	HR (range)	HR (average)	RR (bpm)	BP (systolic)
Preterm	<3	120–160	140	40–60	40–60
Term	3	90–165	125	40–60	60–80
1 month	4	120–180	150	40–60	65–95
6 months	8	110–185	140	25–40	65–105
12 months	10	110–170	135	20–30	70–110
2–3 years	(see formula)	75–150	110	20–30	70–110
4–5 years	(see formula)	65–135	100	20–30	80–110
6–10 years	(see formula)	60–130	90	12–25	90–115
12 years	42	60–120	85	12–25	95–120

Defibrillation and cardioversion basics

The goal of defibrillation and cardioversion is to use external electrical shock to induce coordinated depolarisation of myocardium in order to interrupt a pathologic rhythm and allow sinus node to resume normal pacing.

Synchronised cardioversion delivers an electrical current timed to R or S wave of the intrinsic rhythm, and is intended to convert more organised rhythms such as SVT, atrial fibrillation (AF) or stable ventricular tachycardia (VT). Synchronisation aims to avoid precipitating a disorganised and non-perfusing rhythm such as ventricular fibrillation (VF).

Defibrillation delivers a high-energy, randomly-timed electrical current to convert non-perfusing rhythms: VF and pulseless VT.

A typical unit offers: cardioversion and defibrillation modes, connection to adult or paediatric paddles and/or pads, an oscilloscope to monitor cardiac rhythm, and settings to allow shock at a wide range of energy levels. You must know if your unit is *biphasic* or *monophasic* and how to set it for synchronised shock.

Synchronised cardioversion

See 🕮 Tachycardia p. 90 and AF p. 123 for indications. Remember that a rate <140 is unlikely to be the *cause* of

hypotension.

Technique: patient supine, with IV, O_2, and monitor in place and airway equipment ready. Cardioversion is uncomfortable and patients should be sedated (📖 Procedural anaesthesia and sedation, p. 804).

- Apply paddles (per device instructions or at sternum and right apex) or pads Charge: monophasic: start at 100 J (escalating to 200 J, 300 J, max 360 J). Biphasic: start at 75 J (escalating to 120 J, 150 J, max 200 J). Children: 0.5–1 J/kg (escalating to 2 J/kg) for either biphasic or monophasic units
- 'Clear' anyone in contact with the patient or gurney
- Deliver shock
- Reevaluate rhythm. If successful, consider antiarrhythmic agent to support stable rhythm (e.g. amiodarone). Repeat vitals. If unsuccessful, may escalate voltage

Defibrillation

See 📖 p. 930 for indications.

Technique:

- Apply paddles or pads and turn unit to Defibrillation mode
- Charge: monophasic: 360 J. biphasic: 200 J. Children: 2 J/kg for mono or biphasic
- 'Clear' the patient and deliver shock
- Reevaluate rhythm and continue per resuscitation algorithm

Brain death evaluation

Defined as the irreversible loss of function of the brain, including the brainstem. Diagnosed by clinical parameters; confirmatory tests (angiography, electroencephalography, transcranial Doppler ultrasonography) are optional. Explicit brain death evaluation is usually relevant in settings where mechanical ventilation is available. The determination of clinical brain death should be agreed upon by at least two providers, at least one of them a senior provider.

Diagnostic criteria for clinical diagnosis of brain death

Prerequisites

- Brain death is the absence of clinical brain function when the potential cause is known and demonstrably irreversible
- Clinical or neuroimaging evidence of an acute CNS catastrophe that is compatible with the clinical diagnosis of brain death
- Exclusion of potentially reversible circulatory, metabolic and endocrine disturbances as the cause of the unconsciousness (no shock, and no severe electrolyte, acid-base, or endocrine disturbance)
- No toxicologic explanation such as sedative, hypnotics or narcotics overdose
- Core temperature >36°C (regional variation in threshold, active rewarming may be required prior to evaluation)
- The patient must be apnoeic, requiring ventilatory support

Cardinal findings

Persistence of the three cardinal findings – coma, absence of brainstem reflexes, and apnoea – for 6–24 hours.

Coma (or unresponsiveness)

No cerebral motor response to pain (nail-bed pressure in all extremities and supraorbital pressure).

Absence of brainstem reflexes

- Pupils:
  - » Mid-position (4 mm) to dilated (9 mm)

» No response to bright light (II and III nerves)
- Ocular movement:
  » Doll's eye movement (oculocephalic reflex): rotate the head rapidly to either side (no fracture or instability of the cervical spine). Normally the eye moves opposite to the direction of head movement but in brain death it remains neutral (III and VI nerves)
  » Caloric testing (oculovestibular response): irrigate each ear with 50 ml of cold water (allow 1 minute after injection and at least 5 minutes between testing on each side). Normally the eye moves to opposite side of the test; in brain death there is no such movement (III and VIII nerves)
- Facial sensation and facial motor response:
  » No corneal reflex to touch with a throat swab
  » No jaw reflex
  » No grimacing to deep pressure on nail bed, supraorbital ridge
- Pharyngeal and tracheal reflexes (IX and X nerves):
  » No gag reflex after stimulation of the posterior pharynx with tongue blade
  » No cough response to bronchial suctioning

Apnoea

Absence of spontaneous respiratory movements in the presence of adequate CO_2 drive:

- Prerequisites: core temperature ≥ 36°C, SBP ≥ 90 mm Hg, euvolaemia, normal PCO_2 and normal PO_2
- Procedure: connect a pulse oximeter; disconnect the ventilator; deliver 100% O_2, 6 l/min; look for respiratory movements; measure arterial PO_2, PCO_2, and pH after 5–8 minutes and reconnect the ventilator. Confirm local recommendations for period of apnoea required. Some recommendations suggest up to 10 minutes and require maintenance of SBP > 100 mmHg throughout the trial
- Positive: respiratory movements are absent and arterial PCO_2 ≥ 60 mm Hg
- Negative: respiratory movements are observed

Cranial nerve assessment

There are 12 pairs of cranial nerves: 3–12 have their nuclei in the brain stem.

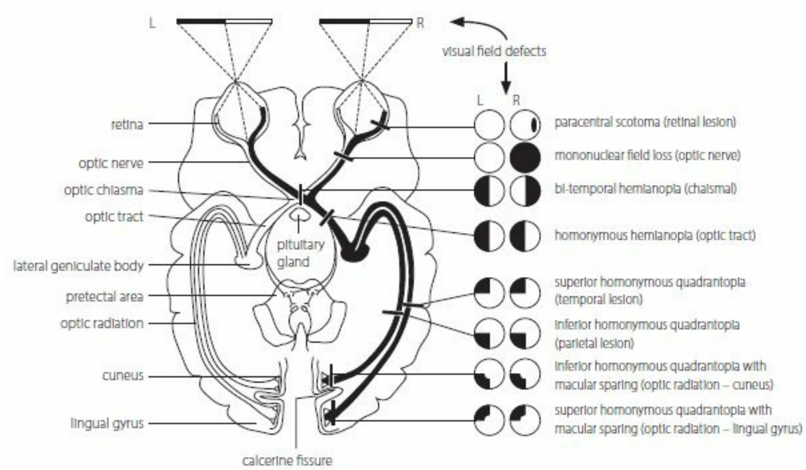
1: Olfactory nerve

- *Brief anatomy*: the olfactory receptors are located on the superior and posterior nasal septum and the lateral wall of the nasal cavity
- Sensory nerve
- Sense of smell usually tested by asking the patient to sniff non-irritating substances (each nostril is tested separately) and to identify the odour

2: Optic nerve

- *Brief anatomy*: from the retina, the optic nerve extends posteriorly through the optic canal to optic chiasm, where the medial fibres cross; pass to the lateral geniculate bodies; give rise to the optic radiations of the occipital cortex
- Sensory nerve
- Examination of the optic nerves includes visual fields, visual acuity, colour detection, pupillary light reaction, and fundoscopic assessment
- Visual fields can be assessed with the confrontation method. Face patient, roughly 0.5 m apart, noses at same level. Close your right eye, while patient closes their left. Keep other eye open and look directly at one another. Move your left arm out and away, keeping it equidistant from the two of you. A raised index finger should be just outside your field of vision. Wiggle finger and bring it in towards your noses. You should both be able to detect it at the same time. Repeat, moving finger in from each direction. Use other hand to check medial field (i.e. starting in front of closed eye). Repeat for other eye. The different patterns of visual field defects have important localisation value.

Figure 1 Patterns of visual field defects



3: Oculomotor nerve

- *Brief anatomy*: located in the superior orbital fissure
- Motor nerve
- Innervates the muscles that perform most eye movements and the muscles of the ciliary body
- See 'testing of cranial nerves 3, 4 and 6' below

4: Trochlear nerve

- *Brief anatomy*: located in the superior orbital fissure
- Motor nerve
- Innervates superior oblique muscle
- See 'testing of cranial nerves 3, 4 and 6' below

6: Abducens nerve

- *Brief anatomy*: located in the superior orbital fissure
- Motor nerve
- Innervates lateral rectus muscle
- See 'testing of cranial nerves 3, 4 and 6' below

Testing of cranial nerves 3, 4 and 6:
- Evaluate all extraocular movements
- Evaluate ptosis
- Evaluate eye position
- Evaluate nystagmus
- Typically done by standing approximately 1 m in front of the patient and having them follow a target without moving their head. Accommodation is tested by moving object towards patient's nose and noting pupillary constriction (📖 p. 509)

5: Trigeminal nerve

- *Brief anatomy*: located in the superior orbital fissure (V1), foramen rotundum (V2) and foramen ovale (V3). Nerves commonly referred to as ophthalmic/V1, maxillary/V2 and mandibular/V3 nerves
- Motor and sensory nerve
- Innervates muscles of mastication and receives sensory input from the face
- Sensation is tested on forehead, cheeks and chin to evaluate three divisions of trigeminal nerve. Muscles of

mastication are tested by asking the patient to clench their jaw

7: Facial nerve

- *Brief anatomy*: located in the internal auditory canal
- Motor and sensory nerve
- Innervates muscles of facial expression. Innervates the stapedius muscle. Sensory fibres get sense of taste from the anterior tongue. Innervates the salivary glands and lacrimal gland
- Inspect muscles of facial expression. Ask patient to:
  - » Raise eyebrows
  - » Close eyes tightly
  - » Smile
  - » Puff out cheeks
  - » Frown
- Sensory fibres can be tested by testing for taste

8: Acoustic nerve

- *Brief anatomy*: located in the internal auditory canal
- Sensory nerve
- Sensory innervation for sound, gravity/equilibrium and rotation
- Sensation is tested by assuring normal auditory acuity. Can be done by rubbing fingers together near ear. Conduct Rinne and Weber test

9: Glossopharyngeal nerve

- *Brief anatomy*: located in the jugular foramen
- Motor and sensory nerve
- Receives taste from posterior part of tongue
- Innervates stylopharyngeus muscle
- Gag reflex tests innervation:
  - » Sound ‘KA’ for palatal innervation
  - » Sound ‘GO’ for articulation

10: Vagus nerve

- *Brief anatomy*: located in the jugular foramen
- Motor and sensory nerve
- Innervation to most laryngeal and pharyngeal muscles – controls muscles for articulation of voice. Provides most parasympathetic nerve fibres to thoracic and abdominal viscera
- Gag reflex tests innervation:
  - » Sound ‘KA’ for palatal innervation
  - » Sound ‘GO’ for articulation

11: Accessory nerve

- *Brief anatomy*: located in the jugular foramen
- Primarily motor nerve
- Innervates the sternocleidomastoid and trapezius muscles
- Test by asking the patient to shrug shoulders

12: Hypoglossal nerve

- *Brief anatomy*: located in hypoglossal canal
- Primarily motor nerve
- Innervates the muscles of the tongue

• Test by asking the patient to move tongue, inspect tongue for atrophy/fasciculations

Glasgow Coma Scale

A 15-point scale for estimating and categorising the severity of traumatic brain injury. Measures the motor, verbal, and eye-opening response. Severity of injury is classified as:
• Mild: GCS 13–15
• Moderate: GCS 9–12
• Severe: GCS 3–8

Component	**Measure**	**Points**
Eye response	Spontaneous eye opening	4
	Opens to verbal command, speech, or shout	3
	Opens to pain, not applied to face	2
	No eye opening	1
Verbal response	Alert and orientated	5
	Confused conversation, but able to answer questions	4
	Inappropriate responses, jumbled phrases, but discernible words	3
	Incomprehensible speech	2
	No sounds	1
Motor response	Obeys commands for movement fully	6
	Localises to noxious stimuli	5
	Withdraws from noxious stimuli	4
	Abnormal flexion, decorticate posturing	3
	Extensor response, decerebrate posturing	2
	No response	1

For infants and small children, use either AVPU or the modified GCS:
• A alert
• V responds to voice (± GCS 13)
• P responds to pain (± GCS 8)
• U unresponsive

Eye opening	**Best motor response**	**Best verbal response**
4 = spontaneously	5 = obeys commands	5 = coos, babbles
3 = to verbal command or shout	4 = localises pain	4 = cries irritably
2 = to pain	3 = flexion to pain	3 = cries to pain
1 = no response	2 = extension to pain	2 = grunts to pain
	1 = no response	1 = no response

Mini mental status exam

The MMSE is the most widely used standardised cognitive screening test; it was intended for use as part of a complete cognitive appraisal and not meant to stand alone as a dementia diagnostic tool. It should be administered by a clinician trained in its use, and takes around 10 minutes to complete (but it is not a timed test).

Instructions for administration and scoring of the MMSE

Orientation (10 points):
a. Ask for the date. Then specifically ask for parts omitted (e.g. ‘Can you also tell me what season it is?’). One point for each correct answer.
b. Ask, ‘Can you tell me the name of this hospital (town, county, etc.)?’ One point for each correct answer.

Registration (3 points):
a. Say the names of three unrelated objects clearly and slowly, allowing approximately one second for each. After

you have said all three, ask the patient to repeat them. The number of objects the patient names correctly upon the first repetition determines the score (0–3). If the patient does not repeat all three objects the first time, continue saying the names until the patient is able to repeat all three items, up to six trials. Record the number of trials it takes for the patient to learn the words. If the patient does not eventually learn all three, recall cannot be meaningfully tested.

b. After completing this task, tell the patient, ‘Try to remember the words, as I will ask for them in a little while’.

Attention and calculation (5 points):

a. Ask the patient to begin with 100 and count backward by sevens. Stop after five subtractions (93, 86, 79, 72, 65). Score the total number of correct answers.

b. If the patient cannot or will not perform the subtraction task, ask the patient to spell the word ‘world’ backwards. The score is the number of letters in correct order (e.g. dlrow = 5, dlorw = 3).

Recall (3 points):

a. Ask the patient if he or she can recall the three words you previously asked him or her to remember. Score the total number of correct answers (0–3).

Language and praxis (9 points):

a. Naming: show the patient a wristwatch and ask the patient what it is. Repeat with a pencil. Score one point for each correct naming (0–2).

b. Repetition: ask the patient to repeat the sentence after you (‘No ifs, ands, or buts’). Allow only one trial. Score 0 or 1.

c. 3-Stage Command: give the patient a piece of blank paper and say, ‘Take this paper in your right hand, fold it in half, and put it on the floor’. Score one point for each part of the command correctly executed.

d. Reading: on a blank piece of paper print the sentence, ‘Close your eyes,’ in letters large enough for the patient to see clearly. Ask the patient to read the sentence and do what it says. Score one point only if the patient actually closes his or her eyes. This is not a test of memory, so you may prompt the patient to ‘do what it says’ after the patient reads the sentence.

e. Writing: give the patient a blank piece of paper and ask him or her to write a sentence for you. Do not dictate a sentence; it should be written spontaneously. The sentence must contain a subject and a verb and make sense. Correct grammar and punctuation are not necessary.

f. Copying: show the patient the picture of two intersecting pentagons and ask the patient to copy the figure exactly as it is. All ten angles must be present and two must intersect to score one point. Ignore tremor and rotation.

Figure 2 Mini-Mental State Examination (MMSE)

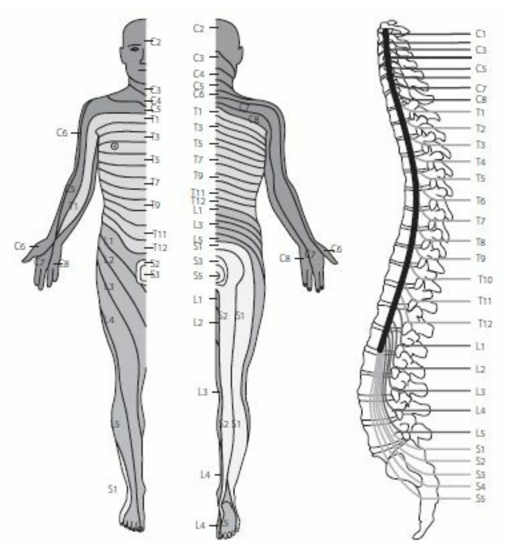
Maximum score	Score	Category
Orientation		
5	( )	What is the (year), (season), (date), (day), (month)
5	( )	Where are we (state), (country), (town or city), (hospital), (floor)
Registration		
3	( )	Name 3 common objects, (e.g. 'apple', 'table', 'penny'). Take 1 second to say each. Then ask the patient to recall all 3 after you have said them. Give 1 point for each correct answer. Then repeat them until he/she learns all 3. Count trials and record. Trails:
Attention and calculation		
5	( )	Spell 'world' backwards. The score is the number of letters in the correct order (D_L_R_O_W_)
Recall		
3	( )	Ask for the 3 objects repeated above. Give 1 point for each correct answer. [Note: recall cannot be tested if all 3 objects were not remembered during registration.]
Language		
2	( )	Name a 'pencil' and 'watch'
1	( )	Repeat the following 'No ifs, ands, or buts'
3	( )	Follow a 3-stage command: 'Take a paper in your right hand, Fold it in half, and Put it on the floor'.
1	( )	Read and obey the following: Close your eyes.
1	( )	Read and obey the following: Write a sentence.
1	( )	Read and obey the following: Copy the following design.

Score ranges

24–30	Normal
18–23	Mild dementia
10–17	Moderate dementia
< 10	Severe dementia

Total score:

Peripheral motor nerves and dermatomes



Upper Limb Myotomes

Shoulder abduction	C5
Elbow flexion	C5, 6
Elbow extension	C7
Wrist extension	C7
Wrist flexion	C8
Finger extension	C7
Finger flexion	C8
Finger abduction	T1

Lower Limb Myotomes

Hip flexion	L1, 2
Hip extension	L5, S1
Knee flexion	L5, S1
Knee extension	L3, 4
Ankle dorsiflexion	L4
Ankle plantarflexion	S1, 2
1st metatarsal extension	L5

Reflexes

Ankle	S1, 2
Knee	L3, 4
Biceps	C5, 6
Triceps	C7, 8

Figure 3 Peripheral motor nerves and dermatomes

Reading the cervical spine X-ray

Anatomy

The axial vertebrae (Figure 4):
- » The first two cervical vertebrae are structurally unique and are designed for rotational motion
- » C1 (atlas) – two lateral masses and an anterior and posterior arch. The lateral masses articulate with the base of the skull
- » C2 (axis) – dens projects superiorly from the anterior arch, and is stabilised against the inner surface of the C1 anterior arch by the transverse ligament

The subaxial vertebrae:
- » C3–7 are structurally similar, and are designed for flexion, extension, lateral flexion, rotation and circumduction
- » Each subaxial vertebra is made up of an anterior vertebral body and a posterior arch, which itself is made up of two pedicles, two laminae, one spinous process, two transverse processes and four articular processes

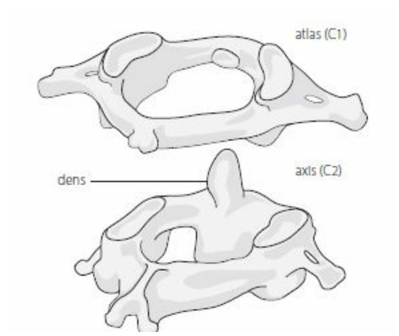


Figure 4 Axial vertebrae

The ligaments (Figure 5):
- » The anterior and posterior longitudinal ligaments run along the along the length of the spine, on either side of the vertebral bodies
- » The ligamentum flavum, supraspinous ligament, interspinous ligament, intertransverse ligament, and capsular ligament stabilise the vertebral arches
- » The intervertebral discs separate the vertebral bodies and act as shock absorbers

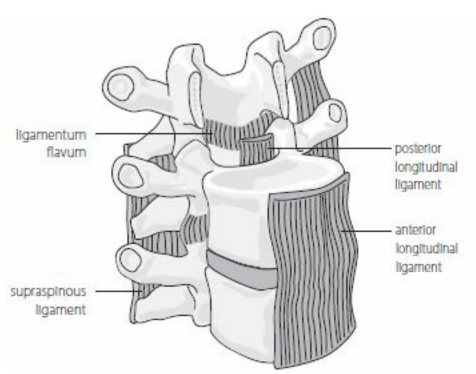


Figure 5 Spinal ligaments

The basics of reading cervical spine X-rays

The standard cervical spine X-ray consists of three images: lateral view, open-mouth odontoid view, and anterior-posterior view. Analyse these views for linear lucencies that may represent fracture, and inconsistencies in

alignment or spacing of the vertebrae.

Lateral view (Figure 6a):

» The lateral cervical view must capture all seven cervical vertebrae, as well as the first thoracic vertebra

» Analyse for four lines: the anterior vertebral line, the posterior vertebral line, the spinolaminar line, and the spinous process line (Figure 4). Any irregularity of these lines suggest a disruption of either the vertebrae themselves or the ligaments

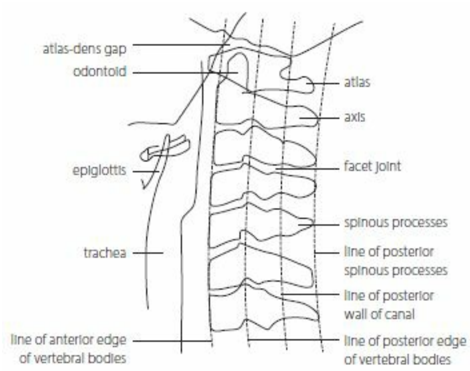


Figure 6a Lateral view

The open-mouth odontoid view (Figure 6b):

» The open-mouth odontoid view isolates C1 and C2 in an anterior-posterior view through the mouth

» Analyse the lateral masses of both the atlas and the axis for alignment. The odontoid should be evaluated for fracture

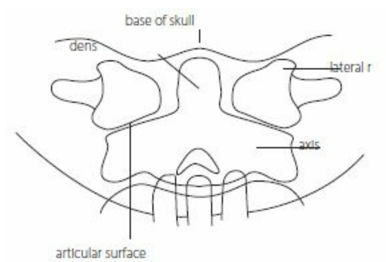


Figure 6b Odontoid peg view

The anterior-posterior view (Figure 6c):

» Analyse the anterior-posterior for alignment of the lateral masses, preserved and consistent disc spaces, and evidence of fractures.

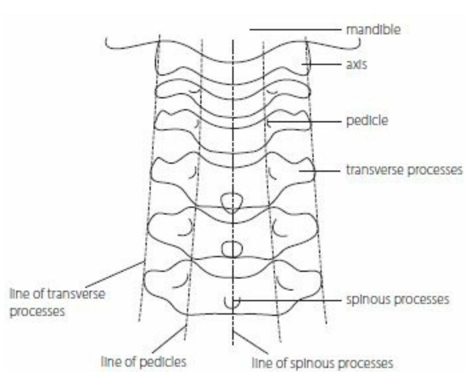


Figure 6c Anterior-posterior view

Common injuries to the cervical spine

Cervical spine injuries are classified as stable or unstable based on the ability of the bony and ligamentous elements to resist displacement and/or spinal cord injury. There are typical patterns of injury that are known to be stable or unstable.

To determine stability the lateral view of the cervical spine is divided into an anterior, middle, and posterior column. Disruption of two or more columns, and/or compression of any vertebrae of > 25%, suggests an unstable injury.

Stable injuries

Odontoid fracture, type I (Figure 7):

» Results from significant, multidirectional force
» Type I odontoid fracture is an avulsion of the superior tip of the odontoid.

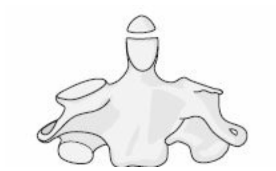


Figure 7 Odontoid fracture, type I

Wedge fracture (Figure 8):

» Results from a flexion-compression force, affected vertebrae is compressed between its superior and inferior neighbours
» Considered stable when compression is < 50% and posterior elements are intact

Figure 8 Cervical wedge fracture

Unstable injuries

Jefferson fracture (Figure 9):

» Results from a direct axial force, which forces the lateral masses of C1 apart.

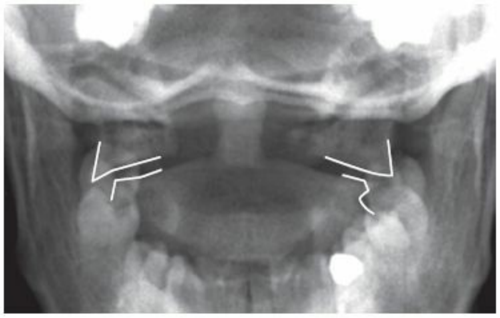


Figure 9 Jefferson fracture

Odontoid fracture, type II and III (Figure 10):
- » Result from significant, multidirectional force
- » Type II odontoid fractures occur at the junction of the odontoid with the body of C2
- » Type III odontoid fractures involve the superior aspect of the anterior arch of C2

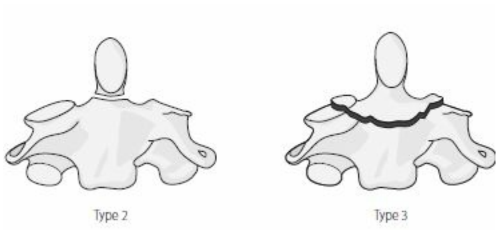


Figure 10 Odontoid fracture, type 2 and type 3

Hangman's fracture (Figure 11):
- » Results from extreme hyperextension, often in deceleration, which cause fractures of the bilateral superior and inferior facets of C2, as well as anterior displacement of C2 upon C3

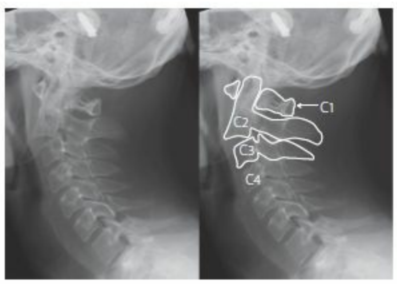


Figure 11 Hangman's fracture

Flexion teardrop fracture (Figure 12):
- » Results from extreme hyperflexion, causing separation of a fragment of the anteroinferior corner of the cervical vertebral, and disruption of ligamentous structures

Figure 12 Flexion teardrop fracture

Bilateral interfacetal dislocation (Figure 13):
» Spontaneous hyperflexion and rotation may allow the articular masses of one vertebra to dislocate over those of the vertebra below
» Will see anterior displacement of the affected vertebra of at least 50% of its width
» By definition, all ligamentous structures are disrupted

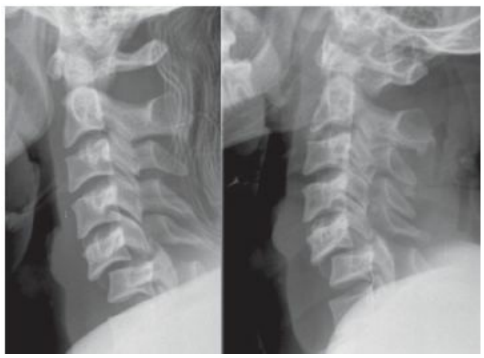


Figure 13 Bilateral interfacetal dislocation

Burst fracture (Figure 14):
» Results from direct axial force
» Multiple vertical fractures of affected vertebral body, causing posterior displacement of a fragment into the spinal cord

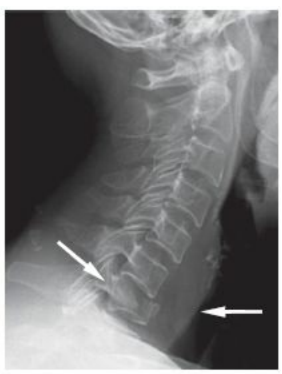


Figure 14 Burst fracture

Reading the chest X-ray

As the most common screening test for pulmonary and cardiac disorders, it is essential to be able to accurately interpret CXR findings. Although experienced providers often read CXRs by gestalt detection, it is important to adopt a systematic approach to maximise accuracy. The history and examination provide the basis for differential diagnoses before the CXR is obtained.

We advocate an ABC method (similar to that taught in the ATLS course).

Technical considerations to first consider when viewing the CXR

A	Anteriorposterior (AP) or posterioranterior (PA) positioning	The heart will appear larger on an AP film
B	Supine or upright	The mediastinum often appears larger on supine films. In the absence of any rotation of the film, the medial ends of each clavicle are equidistant to the spinous process of the vertebra at that level. Positioning has a significant influence on air, fluid and blood vessels in the chest
C	Confirm patient details	Make sure that the X-ray being viewed is from the correct patient
D	Date	Confirm the date on the film
E	Exposure	The end plates of the thoracic vertebra should be just visible through the cardiac shadow
F	Films for comparison	Reviewing old films on the same patient may be helpful

Systematic method of viewing the CXR

A	Airway – large airways, lungs, pleura	Check whether the trachea is midline or deviated
B	Bones – clavicles, ribs, spine	Review bones for fracture or bony destruction. Ribs and intercostal spaces should be symmetrical
C	Cardiac – heart, mediastinum, vascular markings	
D	Diaphragm	Evaluate for evidence of diaphragmatic irregularity or obliteration. The right hemidiaphragm is usually slightly higher than the left
E	Edges – apices, tubes and drains	Evaluate the lung apices for pneumothorax. Make sure tubes are correctly positioned
F	Fields	The lung fields should be assessed for areas of consolidation

Useful signs

- **Silhouette sign**: the loss of a normal lung/soft tissue interface or 'silhouette', caused by any pathology, which either replaces or displaces normal air-filled lung. This sign is commonly applied to heart, mediastinum, chest wall and diaphragm, (e.g. right lower lobe consolidation may obliterate part or all of the right hemidiaphragm, but the right cardiac border would still be clearly defined)
- **Air bronchogram**: bronchi are not normally visible unless seen end-on, or if there is bronchial wall thickening. If alveoli no longer contain air and opacify, the air-filled bronchi passing through the same area may be visible as branching linear lucencies, or air bronchograms
- **Consolidation**: the filling of the alveoli by any cause; pus, blood, fluid. Clinical correlation is needed to make the diagnosis
- **Pleural effusion**: on average, 150 ml must be present for a pleural effusion to be detected on an erect CXR

Reading skull X-rays

Skull X-ray (SXR) is becoming obsolete as it involves significant radiation exposure and is neither sensitive nor specific. In general, SXR is used only when other imaging is not available, and even in these cases, its impact is limited. SXR may contribute to management only in the rare case where it identifies skull fracture that is not clinically evident in a setting where isolated skull fracture would mandate change in management (neurosurgical consult or admission). A normal SXR does NOT rule out skull fracture.

In addition, there are inherent difficulties in reading a SXR, including the similarities between paediatric sutures and fractures, multiple overlying bony structures creating misleading findings, and recurrent artefact effects.

Technique

• Lateral view:
  » Evaluates occipital and frontal bones and the vertex of the skull
• Occipitofrontal view:
  » Evaluates orbits, mandible, zygomatic arches
  » Assure lateral orbital margins are equidistant from lateral aspect of temporal bones
• Towne's view:
  » Evaluates mandibular condyles and the condyle necks
  » Also evaluates middle ear, mastoid sinus, orbital floor

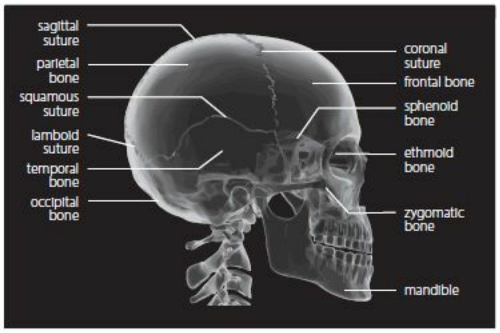
Anatomy evaluated

Pituitary fossa, orbits, temporal bones, temporomandibular junction, mandible, facial bones, sinuses.

Systematic approach

There are many systematic approaches to reading routine radiographic scans of the skull. We recommend addressing each of the following structures in turn:

• Skull:
  » Bony structures
  » Ethmoid bone
  » Frontal bone
  » Occipital bone
  » Parietal bones
  » Sphenoid bone
  » Temporal bone
• Sutures:
  » Coronal
  » Lambdoid
  » Sagittal
  » Squamous
• Soft tissue
• Facial structures
• All sinuses
• Mandible
• Soft tissue structures
• Upper neck
• Cervical spine
• Cartilagenous structures



Reading CT scans of the head

Technique

CT scans are typically done at an angle parallel to the base of the skull; images are usually 5 mm to 10 mm in thickness. Density/tissue attenuation coefficient is measured in Hounsfield Units (HU) between –1 000 (air) and +1 000 (bone). Water is defined at zero Hounsfield Units.

Anatomy evaluated

Cerebral hemispheres, cerebellum, basal ganglia/pons, cisterns, ventricles, bony calvaria.

Systematic approach

There are many systematic approaches to reading routine CT scans of the head. A mnemonic (‘Blood can be very bad’) helps to guide the approach:

B = Blood. Blood typically appears as a hypodense (white) fluid on head CT. It is usually in the range of 50–100 Houndsfield units.

- Epidural haematoma – usually a biconvex, lens shaped collection of blood over the brain convexity
- Subdural haematoma – usually a crescent shaped collection of blood over the brain convexity
- Subarachnoid haemorrhage – blood in any place that is usually occupied by CSF
- Intraparenchymal haemorrhage – blood in the brain parenchyma (trauma, hypertensive haemorrhage)
- Intraventricular haemorrhage – blood in the ventricles

C = Cisterns. Evaluation of the four main cisterns (Sylvian, suprasellar, quadrigeminal and circummesencephalic) for abnormalities. Care must be taken to evaluate for blood, asymmetry and the presence of intracranial pressure/effacement.

B = Brain. Evaluation of brain parenchyma for abnormalities. Care must be taken to evaluate for symmetry, midline shift, differentiation between grey/white matter, mass effect/lesions and changes in density gradients (increased density = blood, calcium, decreased density = ischemia, tumour).

- Evaluate for mass. Typically appear as hypodense, ill-defined lesions on non-contrast CT scans
- Evaluate for infection/abscess. As for mass, will appear as hypodense lesion with surrounding oedema
- Evaluate for stroke. Haemorrhagic = hyperdense, ischaemic = hypodense. Will correlate to location of vessel affected

V = Ventricles. Evaluation of the ventricles for size, symmetry. Increased ventricular size can be seen with increases in ICP. Decreased ventricular size can be seen with overdraining VP shunts.

B = Bone. Evaluation of the bony calvaria for skull fractures.

Blood = blood, Can = cisterns, Be = Brain, Very = ventricles, Bad = bone.

Serum osmolality

Osmolality measures the body’s electrolyte/water balance. It is measured in osmoles – the number of osmoles of solute/kg of solvent.

Normal reference range = 275 to 295 milli-osmoles/kg

Calculated osmolarity = 2 Na + glucose + urea (all in mmol/L)

Osmolar gap = difference between measured and calculated serum osmolality; measures presences of dissolved ions and other molecules dissolved in the serum

The osmolar gap is typically calculated as: measured serum osmolality − calculated osmolality –***a normal osmol gap is < 10 mOsm/kg.***

Unit conversions

Length

UNIT	Multiply By	To find new UNIT
mm	0.04	inches
cm	0.39	inches
m	3.28	feet
m	1.09	yards
km	0.62	miles
inches	25.40	mm
inches	2.54	cm
feet	30.48	cm
yards	0.91	m
miles	1.61	km

Volume

UNIT	Multiply by	To find new UNIT
ml	0.20	teaspoons
ml	0.07	tablespoons
ml	0.03	fluid ounces
Litres	4.23	cups
Litres	2.11	pints
Litres	1.06	quarts
Litres	0.26	gallons
Cubic metres	35.51	cubic feet
Cubic metres	1.31	cubic yards
Teaspoons	4.93	ml
Tablespoons	14.79	ml
Fluid ounces	29.57	ml
Cups	0.24	litres
Pints	0.47	litres
Quarts	0.95	litres
Gallons	3.79	litres
Cubic feet	0.03	cubic metres
Cubic yards	0.76	cubic metres

Weight

UNIT	Multiply by	To find new UNIT
Grams	0.035	ounce
kg	2.20	pounds
Ounces	28.35	grams
Pounds	0.45	kilograms

Temperature

UNIT	Multiply by	To find new UNIT
Fahrenheit	(Temp F – 32) /1.8	Degrees Celsius
Celsius	(Temp C × 1.8) + 32	Degrees Fahrenheit

Visual acuity chart

Visual acuity is measured using a 'Snellen' eye chart. The patient is placed a standard distance (20 feet/6 meters) from the chart and asked to read type print from largest to smallest. Visual acuity is measured by noting the smallest row that can be read accurately.

Snellen chart for adults

Common emergency medications

We have where necessary quoted infusion rates in drops per minute; however, please be aware that the volume (and therefore dose) delivered per drop depends upon the giving set being used.

Hypertensive emergency

In absence of end organ damage do not use IV anti-hypertensives
Always treat pain first, then re-assess BP. Never treat without repeating BP twice
Do not reduce BP more than 25%(📖 p. 106).
See the charts for mixing and administration instructions.

IV nitroglycerin

Dose: 5–300 mcg/min
Start IV 10 mcg/minute, increase by 10 mcg/min every 10 min to 300 mcg/min
Check BP prior to each dose change.

Hydralazine

Dose: 5–10 mg IV or IM repeat in 30 minutes if needed to attain a desired effect
Check BP prior to each dose change.

IV nitroprusside

Avoid in intracranial haemorrhage/increased ICP or renal/liver failure patients.
Dose: 1–10 mcg/kg/min
Start: 1 mcg/kg/min THEN titrate every 5–10 min to desired effect
Check BP prior to each dose change.

IV labetalol

Dose: 10–20mg IV over 2 min then repeat every 10 min. Maximum total dose is 300 mg.
Check BP and HR prior to each dose change.
Always check BP before giving more medications.

Nitroglycerin drip - usual dose range 10–300 mcg/min										
Mix 5 mg in 250 ml of NS (D5 preferable) (concentration 20 mcg/ml)										
mcg/min	5	10	15	20	25	30	35	40	45	50
drops/min	5	10	15	20	25	30	35	40	45	50
drops/sec	0.08	0.17	0.25	0.33	0.42	0.5	0.58	0.67	0.75	0.83
mcg/min	100	150	200	250	300	350	400	450	500	550
drops/min	100	150	200	250	300	350	400	450	500	550
drops/sec	1.67	2.5	3.33	4.17	5	5.83	6.67	7.5	8.33	9.17

**For patients with repeated SBP>160 consider 100–200 mcg (5–10 ml) as a slow IV bolus before starting drip*

mcg/kg/min	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
pt weight	All values below are in drops /min									
5	2	4	6	8	10	12	14	16	18	20
10	4	8	12	16	20	24	28	32	36	40
15	6	12	18	24	30	36	42	48	54	60
20	8	16	24	32	40	48	56	64	72	80
25	10	20	30	40	50	60	70	80	90	100
30	12	24	36	48	60	72	84	96	108	120
35	14	28	42	56	70	84	98	112	126	140
40	16	32	48	64	80	96	112	128	144	160
45	18	36	54	72	90	108	126	144	162	180
50	20	40	60	80	100	120	140	160	180	200
60	24	48	72	96	120	144	168	192	216	240
70	28	56	84	112	140	168	196	224	252	280
80	32	64	96	128	160	192	224	256	288	320
90	36	72	108	144	180	216	252	288	324	360
100	40	80	120	160	200	240	280	320	360	400

Seizures

IV diazepam

Adults: 5–10 mg slow IV push. Repeat up to 3 times if needed.
Children: 0.2 mg/kg (max 10 mg) IV every 5 minutes for 3 doses total. Can be given IM or PR (use the IV solution PR at 0.5 mg/kg, not to exceed 10 mg).
If no response after 3 doses of diazepam, proceed to phenobarbitone.

IV phenobarbitone

Dose: 10–20 mg/kg IV slowly (NOT MORE THAN 50 mg/min in adults, and 30 mg/min in paediatrics); repeat every 20 minutes up to 30 mg/kg total.
Caution: risk of respiratory depression with phenobarbitone is high especially when given after diazepam - monitor HR, BP and especially RR during infusion.

IV phenytoin

Dose: 15–20 mg/kg IV slowly (NOT MORE THAN 50 mg/min in adults, and 1 mg/kg/min in paediatrics not exceeding 50 mg/min).
Caution: drops BP if given too fast: slow the infusion rate or stop. Always check RR, HR, and BP when infusing phenytoin.

Acute asthma

For all adult and paediatric moderate to severe asthma:

Nebulise

And repeat as needed – deliver continuous if severe:
Salbutamol 10 mg (adults) or 5 mg (children)

Plus
Ipratropium bromide 500 mcg (1 ampoule)
Plus
Normal saline (equal volume)

IV hydrocortisone

Adults: 200 mg IV as a single dose
Children: 2 mg/kg IV as a single dose
Plus
If the patient can take orally, oral prednisone 60 mg stat for adults and 2 mg/kg for paediatrics (not to exceed 60 mg)

IV dexamethasone

Adults: 20 mg IV as a single dose
Children: 0.5 mg/kg IV as a single dose, not to exceed 20 mg
If patient is not improving with the above therapies, give adrenaline.

Adrenaline

If no IV access:
Adults: 0.3 mg IM (front of thigh) every 5 minutes
Children: 0.15 mg IM (front of thigh) every 5 minutes
For IV drip:
1 mg of adrenaline in 500 ml of NS. Start the drip at 1 DROP per SECOND and titrate to patient response as needed.

Magnesium sulphate

Adults: 2 g IM (front of thigh) stat
Children: 50 mg/kg (max 2 g) IM (front of thigh) stat
If intubating for asthma use 2 mg/kg of IV ketamine as induction agent because ketamine does NOT suppress respiratory drive.

Rapid sequence intubation

A combination of sedation and muscle relaxation.

Sedatives

Etomidate

Dose: 0.3 mg/kg (0.15 mg/kg if hypotensive) IV; not to exceed 20 mg

Ketamine

Dose: 2 mg/kg IV once
Ketamine is the preferred agent for intubation for asthma and anaphylaxis.

Midazolam

Dose: 0.2 mg/kg IV once
Intubation doses are much higher than sedation doses (15–20 mg).

Diazepam

Dose: 0.3 mg/kg IV once

Paralytics

Always give sedation before paralytics.

Succinylcholine

Dose: 2 mg/kg IV once
Onset of action: 15–30 seconds
Duration of action: 10–20 minutes
May cause hyperkalaemia. Caution in renal failure or patients with muscular diseases and cachexia.

Pancuronium

Patients will be paralysed for more than an hour. Provide adequate sedation after intubation.

Post intubation sedation

Give pain medication first:

Morphine

Dose: 0.05–0.1 mg/kg IV every 30 minutes as needed, do not exceed 10 mg per dose (can cause low BP)

Pethidine

Dose: 1 mg/kg IV every 4 hours as needed
Then give sedation medication:

Midazolam

Dose: 0.05–0.1 mg/kg IV every 30 minutes as needed (usual starting dose: 2 mg)

Diazepam

Dose: 0.05–0.2 mg/kg IV every 60 minutes as needed (usual starting dose 5–10 mg)

Inotropes and chronotropes

For adults and children

Dopamine

Dose: 1–20 mcg/kg/min:

Dopamine drip – usual dose range 1–20 mcg/kg/min										
Mix 200 mg in 500 ml of NS (concentration 400 mcg/ml)										
mcg/min	**50**	**75**	**100**	**125**	**150**	**200**	**250**	**300**	**350**	**400**
drops/min	2.5	3.75	5	6.25	7.5	10	12.5	15	17.5	20
drops/sec	0.04	0.06	0.08	0.1	0.13	0.17	0.21	0.25	0.3	0.33
mcg/min	**500**	**600**	**700**	**800**	**900**	**1000**	**1250**	**1500**	**1750**	**2000**
drops/min	25	30	35	40	45	50	62.5	75	87.5	100
drops/sec	0.4	0.5	0.6	0.7	0.75	0.8	1.0	1.3	1.5	1.7

Dobutamine

Dose: 1–20 mcg/kg/min

Dobutamine drip – usual dose range 1–20 mcg/kg/min										
Mix 200 mg in 500 ml of NS (concentration 400 mcg/ml)										
mcg/min	**50**	**75**	**100**	**125**	**150**	**200**	**250**	**300**	**350**	**400**
drops/min	2.5	3.75	5	6.25	7.5	10	12.5	15	17.5	20
drops/sec	0.04	0.06	0.08	0.1	0.13	0.17	0.21	0.25	0.3	0.33
mcg/min	**500**	**600**	**700**	**800**	**900**	**1000**	**1250**	**1500**	**1750**	**2000**
drops/min	25	30	35	40	45	50	62.5	75	87.5	100
drops/sec	0.4	0.5	0.6	0.7	0.75	0.8	1.0	1.3	1.5	1.7

Adrenaline

Dose: 0.05–0.5 mcg/kg/min

Adrenaline drip – usual dose range 0.05–0.5 mcg/kg/min										
Mix 1 mg in 500 ml of NS (concentration 2mcg/ml)										
mcg/min	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
drops/min	10	20	30	40	50	60	70	80	90	100
drops/sec	0.2	0.3	0.5	0.7	0.8	1.0	1.2	1.3	1.5	1.7
mcg/min	**15**	**20**	**25**	**30**	**35**	**40**	**50**	**60**	**70**	**80**
drops/min	150	200	250	300	350	400	500	600	700	800
drops/sec	2.5	3.3	4.2	5.0	5.8	6.7	8.3	10.0	11.7	13.3

Hypoglycaemia

All children up to 40 kg	Adults and children > 40 kg
Dilution to 10% solution preferred in children	2 ml/kg of 50% dextrose OR 4 ml/kg of 25% dextrose
5 ml/kg of 10% dextrose	5–10 ml/kg of 10% dextrose

If no other dextrose is available, give 20 ml/kg boluses of IV dextrose 5%.
Re-assess blood glucose 30 minutes after every bolus.

Hypokalaemia

Severe hypokalaemia (potassium < 2.5 mEq/L)

Adults

PO: 40–60 mEq every 2 hours for 2–3 doses
IV: 10 mEq/hour via peripheral line for 6 hours

Children

PO: 1 mEq/kg every 2 hours for 2–3 doses
IV: 1 mEq/kg dose. Give doses below 10 mEq over one hour. For higher doses, do not exceed 10 mEq per hour infusion via peripheral line. Reassess the potassium level after dose.

Mild to moderate (potassium 2.5–3.5 mEq/L)

Give PO supplements.
Adults: 40 mEq orally once
Children: 0.5–1 meq/kg orally once.

Hyperkalaemia

For symptomatic patients (ECG changes) with potassium > 6 mEq/l, begin treatment IMMEDIATELY and repeat serum potassium.

Calcium gluconate

Adults: 1 gram IV as a slow push (over 5–10 minutes)
Children: 100 mg/kg/dose; not to exceed 1 gram IV as a slow push (over 5–10 minutes)
OR

Calcium chloride

Reserve for critical patients with life threatening situations.
Adults: 1 gram IV as a slow push (over 5–10 minutes)
Children: 20 mg/kg/dose; not to exceed 1 gram IV as a slow push (over 5–10 minutes)

Dextrose and insulin

All children up to 40 kg	**Adults and children > 40 kg**
5 ml/kg of 10% dextrose	2 ml/kg of 50% dextrose
	4 ml/kg of 25% dextrose

Check blood glucose prior if possible. Do NOT give any dextrose if blood glucose > 14 mmol/L.
Adults: 10 units of regular (soluble) insulin as IV push
Children: 0.1 units/kg of regular (soluble) insulin as IV push
Reduce insulin dose by 50% in renal failure patients.
Must do serial RBG every 1 hour.

B_2-agonists. Salbutamol nebuliser.

Adults and children: nebulise the patient with 10 mg of salbutamol

IV furosemide

Adults: 40 mg IV once
Children: 1 mg/kg IV once, not to exceed 40 mg

Sodium bicarbonate

Adults: 50 mEq IV once
Children: 1 mEq/kg IV once, not to exceed 50 mEq

Anticoagulant, antiplatelet and thrombolytic medications

Institutional protocols should always be consulted and the risk of bleeding/thrombosis taken into consideration before initiating any of these agents.

Drug	Targets	Route	Daily dosing	Laboratory monitoring	Reversal for life-threatening bleeding	Notes
Warfarin (vitamin-K antagonists)	Factors II, VII, IX, X, and protein C&S	PO	Once	INR	2–4 units FFP OR 25–50 units/kg 3 or 4 factor PCC	Rapid reversal can only be achieved with the use of FFP or PCC in combination with Vitamin K 5–10 mg IV 15–30 min infusion
Heparin	Factor IIa, Xa, and other non-specific targets	IV SQ	IV: continuous SQ: 2–3 times	aPTT, PT, INR, platelets	1 mg of protamine for every 100 units of heparin (max 50 mg)	For continuous infusion, for reversal multiply the hourly rate by 2–3 to calculate protamine dose
Low molecular weight heparins (e.g. enoxaparin)	IIa, Xa	IV SQ	Once to twice	Renal function, anti-Xa levels	1 mg of protamine neutralises 1mg of enoxaparin	Protamine only neutralises 60–75% of LMWH activity
Direct thrombin inhibitors	Thrombin	PO IV SQ	PO: Once to twice IV: continuous	aPTT, ACT, Hb; oral regimens do not require monitoring	Unknown Consider: charcoal for oral ingestion; haemodialysis	Safe for patients with history of heparin-induced thrombocytopenia. Caution in renal dysfunction
Xa inhibitors	Xa	PO SQ	Once to twice	Renal function, CBC, platelets	Unknown Consider charcoal for oral ingestion.	Xa agents are not dialysable
Aspirin	COX enzymes	PO	Once	CBC, platelets	Consider platelet transfusion	Restoration of normal platelet function occurs few days after discontinuation
Clopidogrel	P2Y12 ADP receptor	PO	Once	CBC, platelets	Consider platelet transfusion	Restoration of normal platelet function occurs few days after discontinuation
Streptokinase tPA	Fibrin-specific plasminogen	IV	One-time dose	Platelets, INR, PTT	None	Usual indications: STEMI, massive PE

<table>
<tr><th>Drug</th><th>Dosing in emergency use</th><th>Notes</th></tr>
<tr><td>Warfarin</td><td>Patient-specific
Usual starting dose: 5 mg PO at bedtime</td><td>Requires frequent monitoring. Educate about drug and food interactions</td></tr>
<tr><td>Heparin</td><td>ACS:
60 units/kg IV as the initial bolus (max 4 000 units), followed by 12 units/kg/hour (max 1 000 units/hour)

PE or venous thrombosis (VT):
80 units/kg IV as the initial bolus (max 10 000 units), followed by 18 units/kg/hour</td><td>Example titration instructions (aPTT check q6 hours):
< 40 seconds: repeat bolus
40–45.9 seconds: increase infusion by 200 units/hour
46–52.9 seconds: increase infusion by 100 units/hour
53–74.9 seconds (THERAPEUTIC): recheck aPTT in 6 hours, if therapeutic recheck aPTT daily
75–85.9 seconds: decrease infusion by 100 units/hour
86–94.9 seconds: hold infusion for 30 minutes, decrease by 200 units/hour
> 95 seconds: hold infusion for 1 hour, decrease by 300 units/hour</td></tr>
<tr><td>LMWH</td><td>Enoxaparin for ACS or PE/VT
1 mg/kg SQ twice daily, or 1.5 mg/kg SQ daily in admitted patients</td><td>Enoxaparin should be avoided in patients with CrCl < 30
<table>
<tr><th>Weight (kg)</th><th>1 mg/kg</th><th>1.5 mg/kg</th></tr>
<tr><td>35– 40</td><td>40 mg</td><td>60 mg</td></tr>
<tr><td>50– 69</td><td>60 mg</td><td>80 mg</td></tr>
<tr><td>70– 89</td><td>80 mg</td><td>120 mg</td></tr>
<tr><td>90–109</td><td>100 mg</td><td>150 mg</td></tr>
<tr><td>110–130</td><td>120 mg</td><td>180 mg</td></tr>
</table></td></tr>
<tr><td>Direct thrombin inhibitors</td><td>Dabigatran for AF 150 mg PO BID</td><td>No specific antidote exists.</td></tr>
<tr><td>Xa inhibitors</td><td>Rivaroxaban for AF, PE and VT: 20 mg PO daily
Rivaroxaban for knee/hip replacement VT prophylaxis: 10 mg PO daily 2–4 weeks</td><td>Other Xa inhibitors:
Apixaban (PO) for AF, PE, and VT:
2.5–5 mg by mouth twice daily
Fondaparinux (injectable) is a Xa inhibitor with similar indications to enoxaparin</td></tr>
<tr><td>Aspirin</td><td>ACS
150–325 mg chewed as soon as possible</td><td>Contraindicated only in true anaphylaxis or active gastrointestinal bleeding</td></tr>
<tr><td>Clopidogrel</td><td>ACS
300 mg PO as soon as possible</td><td>May be used in patients with true allergy to aspirin as a substitute</td></tr>
<tr><td>Strepotokinase, tPA</td><td>Acute STEMI
SK: 1.5 million units over 1 hour
tPA: 15 mg IV push, followed by 0.75 mg/kg (max 50 mg) over 30 min, followed by 0.5 mg/kg (max 35 mg) over 1 hour</td><td>We do not recommend thrombolysis for ischaemic stroke; if used, no evidence for SK
tPA: 0.9 mg/kg (max 90 mg). 10% of dose to be administered as an IVP, with the remaining 90% administered over 1 hour
Massive PE
tPA: 100 mg IV over 2 hours (or IV push if cardiac arrest)
SK: 250 000 U loading over 30 mins, then 100 000 U/hour for 24 hours</td></tr>
</table>

Antibiotic guidelines

Meningitis

Symptoms less pronounced in immunosuppressed, elderly, HIV+, or children > 1 year. May be difficult to differentiate bacterial from viral, tubercular, cryptococcal, fungal or aseptic in these patients. CSF results may be suggestive. If CSF is unavailable or not confirmatory, initiate broad treatment with all possible antimicrobials, narrowing therapies as cultures and other studies result.

Acute bacterial meningitis: neonates *Bacterial pathogens in first month of life include Strep agalactiae, E. coli, Listeria, Klebsiella.*	≥7 days Ampicillin 50 mg/kg IV TID AND gentamicin 5 mg/kg once QD 8–28 days Ampicillin 50 mg/kg IV Q6h AND gentamicin 7.5 mg/kg once QD ADD cefotaxime 75mg/kg IV TID if suspect gram-negative organism
Acute bacterial meningitis: 1 month–5 years *Bacterial pathogens include Strep pneumoniae, Neisseria meningitides, Strep agalactiae, Haemophilus influenzae, E. coli. Cover gram-negative organisms, including Pseudomonas, and Staphylococcus in recent CNS surgical procedures or trauma (e.g. skull fracture, CSF shunt, neurosurgery)*	Preferred base regimen 29 days–3 months Ampicillin 50 mg/kg (max 2 g) IV Q6h AND Ceftriaxone 50 mg/kg (max 2 g) IV BID 3 months–5 years Ceftriaxone 50 mg/kg (max 2 g) IV BID Alternate base regimen Chloramphenicol 25 mg/kg (max 1g) IV Q6h AND [Benzylpenicillin 100 000 units/kg (max 4 million units) IV Q4h OR Ampicillin 50 mg/kg (max 2 g) IV Q6h]
	Additional agents by risk – ADD to base regimen as needed *Possible MRSA or penicillin-resistant Strep pneumoniae* ADD vancomycin 15 mg/kg IV Q6h *Possible MSSA* ADD cloxacillin 50 mg/kg (max 2 g) IV Q4h *Possible pseudomonas* SUBSTITUTE for ceftriaxone: cefepime 50 mg/kg (max 2 g) IV TID OR meropenem 40 mg/kg (max 2 g) IV TID
Acute bacterial meningitis: children > 5yrs and adults ***Bacterial pathogens include Neisseria meningococcus, Streptococcus pneumoniae, Haemophilis influenzae. For age over 50yrs, Listeria, gram-negative bacilli***	Preferred base regimen Ceftriaxone 2 g IV BID Alternate base regimen Chloramphenicol 1–1.5 g IV Q6h AND [Benzylpenicillin 4 million units IV Q4h OR ampicillin 2 g IV Q4h] *Pen-allergic* Vancomycin 15 mg/kg IV TID (child: 15 mg/kg IV Q6h) AND Moxifloxacin 400 mg IV QD

	Additional agents by risk – ADD to base regimen as needed *Adults > 50 yrs or immunocompromise* ADD ampicillin 2 g IV Q4h to regimen Pen-allergic: co-trimoxazole (TMP/SMX) 5 mg/kg/TMP IV Q6h *Possible MRSA or penicillin-resistant Strep pneumo* ADD vancomycin 15 mg/kg IV TID (child: 15 mg/kg IV Q6h) *Possible MSSA* ADD cloxacillin 2 g IV Q4h *Possible pseudomonas* SUBSTITUTE for ceftriaxone: cefepime 2 g IV TID OR meropenem 2 g IV TID
Meningitis: immunosuppression (HIV, malnutrition, chemotherapy, chronic steroid use), recent CNS procedure or trauma *Coverage includes Klebsiella, Pseudomonas, MRSA, Listeria. Always consider TB in immunocompromise*	Cefepime 2 g IV TID (child: 50 mg/kg IV TID) OR meropenem 2 g IV TID (child: 40 mg/kg IV TID)] AND Ampicillin 2 g IV Q4h (child: 50 mg/kg IV Q4h) AND [Vancomycin 15 mg/kg IV TID (child: 15 mg/kg IV Q6h (MRSA) OR cloxacillin 2 gm IV Q4h (child: 50 mg/kg IV Q4h) (MSSA)]
Cryptococcal meningitis *Given relatively limited side effects of fluconazole, even at high-doses, and 100% mortality of untreated or inappropriately treated cryptococcal meningitis, expert recommendations and literature support high-dose fluconazole therapy when other agents unavailable*	Preferred regimen Induction: amphotericin B 0.7 mg/kg IV QD + flucytosine 25 mg/kg PO Q6h × 2 weeks minimum Consolidation: fluconazole 400 mg PO QD × 8 weeks minimum Alternate regimen Induction: amphotericin B 0.7 mg/kg IV QD + fluconazole 800 mg PO QD × 2 weeks minimum Consolidation: fluconazole 800 mg PO QD × 8 weeks minimum Other agents unavailable Induction: fluconazole 1200–2000 mg PO QD × 10–12 weeks minimum Consolidation: fluconazole 800 mg PO QD × 8 weeks minimum ** Do not transition from induction to consolidation until CSF is sterile and patient clinically improved Maintenance therapy Fluconazole 200 mg PO QD for life Management symptomatic increased ICP during induction If symptoms of increased ICP, obtain CT head to rule out mass lesion. If no mass lesion, LP. If opening pressure on LP > 25 cm, drain CSF to reduce pressure by 50% of opening pressure or to < 20 cm, whichever is higher (e.g. opening pressure of 80 cm gets drained by half to 40 cm. OP 30 cm gets drained to 20 cm). Repeat QD until opening pressure consistently at or below 25 cm and symptoms resolved. If symptoms of increased ICP return, repeat LP, check pressure, consider IRIS vs refractory crypto. Infusion reactions common with amphotericin. Consider pre-treatment with antipyretics (paracetamol), antihistamine (chlorphenamine). Pre-treat with IVF to protect kidneys. No benefit mannitol, acetazolamide, steroids for increased ICP. ARV therapy should be initiated approximately 2 weeks after initiation of cryptococcal meningitis treatment; earlier initiation of ARVs associated with increased mortality.
CNS TB, no suspected resistance *See steroids below for adjunct steroids in CNS meningitis and dosing protocols.* *Reference national TB guidelines given varying TB sensitivities and regimens* *Always administer anti-TB in co-operation with local tuberculosis treatment agencies*	Meningitis: four-drug regimen Isoniazid AND rifampin AND pyrazinamide AND [ethambutol OR streptomycin] × 2 months. Then, isoniazid AND rifampin × 7–10 months. If pyrazinamide not available, extend treatment to 18 months Tuberculoma: four-drug regimen as above, with INH + RIF × 16 months, for total 18-month treatment Dosing: Isoniazid 15 mg/kg (max 300 mg) Rifampin 10–200 mg/kg/day (max 600 mg) Pyrazinamide 15–30 mg/kg/day (max 2 g) Streptomycin 20–40 mg/kg/day (max 1 g) Ethambutol 15–25 mg/kg/d (max 1 g)
Viral *Consider tick fever or fungal meningitis as other infectious aetiologies aseptic meningitis*	Herpes simplex Neonatal: acyclovir 20 mg/kg IV TID × 21 days Children: acyclovir 10–15 mg/kg IV TID × 14–21 days > 11 years: acyclovir 10 mg/kg IV TID × 14–21 days
Steroids in meningitis *Literature suggests that steroids may improve outcomes in S. pneumo meningitis presenting early, but there are no Africa-based studies showing benefit. We do not recommend early empiric steroids for suspected bacterial or viral meningitis*	TB meningitis: literature suggests improved outcomes in both adults and children Dexamethasone regimen: < 25 kg: 8 mg QD × 2 weeks, then 6 week taper > 25 kg: 0.3–0.4 mg/kg QD × 2 weeks, then 0.2 mg/kg QD × 2 weeks, then 4 mg/day × 1 week, then 3 mg/day × 1 week, then 2 mg/day × 1 week, then 1 mg/day × 1 week Prednisolone regimen: 2–4 mg/kg PO QD (max 60 mg) × 2 weeks, followed by a slow tapering dose over 6 weeks, for total 8 weeks therapy
Meningococcemia epidemic	Ceftriaxone 100 mg/kg/day IM (max 4 g/day) × 1 dose; repeat × 1 on 2nd day if no improvement Oily chloramphenicol 100 mg/kg/day IM (max 4 g/day) × 1 dose; repeat × 1 on 2nd day if no improvement‡
Exposure prophylaxis	See 📖 Meningitis p. 330

Pneumonia

Comorbidities include CCF, renal disease, liver disease, malignancy, history of stroke, neonate, elderly, immunocompromise, malnutrition, alcoholism, diabetes, sickle cell.

Adults

Example respiratory antibiotics by class

β-lactams	Macrolides
Ceftriaxone 1 g IV QD	Azithromycin 1 g once, then 500 mg IV/PO QD
Cefotaxime 2 g IV TID	Erythromycin 500 mg PO Q6h
Ampicillin-sulbactam 3 g IV Q6h	Doxycycline 100 mg PO BID may be used as alternate
Pen-allergic: aztreonam 2 g IV TID	Respiratory fluoroquinolones *
Anti-pseudomonal β-lactams	Levofloxacin 750 mg PO/IV QD
Cefepime 2 g IV TID	Moxifloxacin 400 mg PO/IV QD
Meropenem 1 g IV QD TID	
Piperacillin-tazobactam 4.5 g IV Q6h	

Pneumonia	
Community-acquired pneumonia, out-patient treatment *Well appearing with no immunocompromise, no comorbidities, no signs of shock or respiratory distress, able to take PO*	Preferred: Macrolide × 5 days Alternate: Doxycycline × 7–10 days
Community-acquired pneumonia, in-patient treatment *Mildly ill-appearing or mild respiratory distress or significant comorbidity*	[β-lactam AND macrolide] OR Respiratory fluoroquinolone
Severe pneumonia	
Severe pneumonia *Shock, severe respiratory distress, hospital-acquired, multiple comorbidities*	Base regimen β-lactam AND [Respiratory fluoroquinolone OR azithromycin] *Pen-allergy* Aztreonam AND respiratory fluoroquinolone *If limited antibiotics available*, chloramphenicol 1 g IV Q6h
	Additional agents by risk ADD vancomycin or clindamycin if possible MRSA (see below) ADD cloxacillin if possible MSSA (see below) ADD anaerobic coverage if possible aspiration (see below) See pseudomonas and cryptococcus regimens below
Organism-specific coverage	
Staph coverage *Pustular lesions, indwelling line, soft tissue infection, air-fluid levels or abscess, IV drug use, hospitalisation, severe illness*	*MRSA* Vancomycin 15–20 mg/kg IV BID If vancomycin unavailable: clindamycin 600 mg IV TID *MSSA* Cloxacillin 2 g IV Q6h

Aspiration/anaerobic coverage *Alcoholics, hospitalised, bed-bound, NG tubes, witnessed aspiration*	Clindamycin 600 mg IV TID OR Metronidazole 500 mg IV/PO TID
Pseudomonas *Severe immunosuppression, bronchiectasis, history of pseudomonas*	Anti-pseudomonal β-lactam (aztreonam if pen-allergic) AND Gentamicin 7 mg/kg IV QD AND [Azithromycin OR anti-pseudomonal fluoroquinolone (ciprofloxacin 400 mg IV TID, levofloxacin 750 mg IV QD)]
Cryptococcus *Mild disease presumes no immunosuppression, no pulmonary infiltrates CSF negative for dissemination*	Mild disease: fluconazole 400 mg PO QD × 6–12 months, followed by at least 6–12 months maintenance Moderate to severe disease: treatment as for cryptococcal meningitis; see 🕮 Meningitis antibiotics
HIV *Extensive list of organisms causing pulmonary symptoms in HIV patients. An approach to emergency department empiric management listed; further treatment dependent upon patient risk factors, imaging, cultures, response to treatment*	*Treat bacterial causes* See severe pneumonia regimens above *PJP pneumonia* First line: co-trimoxazole (320 mg TMP) PO TID Severe disease: co-trimoxazole (5 mg/kg TMP) IV TID Alternate: pentamidine 4 mg/kg/day IV once QD Prednisone 40 mg PO BID followed by 2 week taper for severe hypoxia or respiratory distress If failing on oral regimen, switch to IV. If no improvement within 5 days, switch to alternate drug *Consider TB in any HIV+ patient with respiratory symptoms* For subacute course not responding to routine management, *consider fungal aetiologies*, including histoplasmosis, cryptococcus
Children	
Neonatal (< 1 month)	*0–7 days* Ampicillin 50 mg/kg IV/IM TID + gentamicin 5 mg/kg IV/IM QD *8–28 days* Ampicillin 50 mg/kg IV/IM Q6h + gentamicin 7.5 mg/kg IV/IM QD
Pneumonia *Cough or difficulty breathing PLUS fast breathing or chest indrawing (RR > 50/min in age 2–11 mos; RR> 40/min in 1–5yrs)*	Amoxicillin 40 mg/kg (max 2 g) PO BID × 5 days minimum
Severe pneumonia *Cough or difficulty breathing PLUS central cyanosis, O2 sat <90%, severe respiratory distress, lethargy, unconsciousness, convulsions, inability to suck or drink (WHO)* *Malnutrition, immunosuppression*	Base regimen Ampicillin 50 mg/kg IV/IM Q6h × 5 days AND Gentamicin 7.5 mg/kg IV/IM QD Alternate: ceftriaxone 50 mg/kg IV BID AND gentamicin *Pen-allergy* Levofloxacin 10 mg/kg IV/PO BID (see notes below for risk/benefit fluoroquinolone use in children) *If limited antibiotic options*, chloramphenicol 25 mg/kg IV Q6h

	Additional agents by risk ADD vancomycin 15 mg/kg IV Q6h if concern for MRSA; clindamycin 10 mg/kg IV/PO if no MRSA resistance or vancomycin unavailable ADD cloxacillin 50 mg/kg IV Q6h if concern for MSSA ADD azithromycin 10 mg/k IV × 2 days, then 5 mg/kg thereafter OR erythromycin 10 mg/kg IV Q6h if atypical pneumonia
HIV	*Treat bacterial causes* See Severe pneumonia above *PJP pneumonia* Children < 12mos: treat empirically for PCP as below Children 12–59mos: treat only if signs PCP First line: co-trimoxazole (8 mg/kg TMP) IV/PO TID × 3 weeks (IV preferred) Alternate: pentamidine 4 mg/kg IV QD × 3 weeks Add prednisolone 1–2 mg/kg QD for severe hypoxia or respiratory distress, followed by 2 week taper for severe hypoxia or respiratory distress If failing on oral regimen, switch to IV. If no improvement within 5 days, switch to alternate drug *Consider TB in any HIV+ patient with respiratory symptoms* For sub-acute course not responding to routine management, consider fungal aetiologies *Cryptococcus* Mild: fluconazole 6–12mg/kg (max 400 mg) PO QD × 6–12 months Mod-severe: treat as CNS. See Cryptococcal meningitis antibiotic regimens
Severe infection without focus and sepsis	
Neonate	*0–7 days* Ampicillin 50 mg/kg IV TID AND gentamicin 5 mg/kg once QD *8–28 days* Ampicillin 50 mg/kg IV Q6h AND gentamicin 7.5 mg/kg once QD *Suspected Staph infection* SUBSTITUTE cloxacillin 50 mg/kg IV BID (≥ 7days) or TID (8–28 days) for ampicillin (MSSA) OR ADD vancomycin 10 mg/kg IV BID (≥ 7days) or TID (8–28 days) (MRSA)
Child	*Sepsis of unknown aetiology* Cloxacillin 50 mg/kg IV q4h AND ceftriaxone 50 mg/kg IV BID *Possible respiratory source* ADD gentamicin 7.5 mg/kg IV QD *Possible abdominal or pelvic source* ADD metronidazole 12.5 mg/kg IV TID *Possible MRSA* SUBSTITUTE vancomycin 10–15mg/kg IV Q6h for cloxacillin

Adult	*Sepsis of unknown aetiology* Cloxacillin 2 g IV q4h AND Ceftriaxone 1 g IV QD AND Metronidazole 500 mg IV TID *Possible MRSA* SUBSTITUTE vancomycin 15–20 mg/kg IV BID for cloxacillin *Strongly suspected abdominal or pelvic source* [Ceftriaxone AND metronidazole] OR [Ciprofloxacin 400 mg IV BID AND metronidazole]
Neutropaenia *Avoid chloramphenicol in neutropaenia; suppresses cell lines*	Double gram-negative and anti-Pseudomonal coverage [Cefepime 2 g IV TID OR meropenem 1 g IV TID] AND [Ciprofloxacin 400 mg IV BID OR gentamicin 7 mg/kg IV QD] ADD vancomycin if concern for MRSA ADD cloxacillin if concern for MSSA
Alternative empiric antibiotic regimens *Penicillin allergy, other agents unavailable*	*Alternate* Cloxacillin 2 g IV Q4h (child: 50 mg/kg IV Q4h) AND Chloramphenicol 1 g IV Q6h (child: 25 mg/kg IV Q6h) AND Gentamicin 7 mg/kg IV QD *Alternate (no staph coverage)* Ampicillin 2 g IV Q4h (child: 50 mg/kg IV Q4h) AND Gentamicin 7 mg/kg IV QD AND Metronidazole 500 mg IV TID (child: 12.5 mg/kg IV TID) *Pen-allergy* [Clindamycin 600 mg IV TID (child: 10 mg/kg IV Q6h) AND Ciprofloxacin 400 mg IV BID (child: 15 mg/kg IV BID)] OR [Vancomycin 20 mg/kg IV BID (child: 10–15 mg/kg IV Q6h) AND Aztreonam 2 g IV TID (child: 30 mg/kg IV TID)]

Skin and soft tissue infections

Soft tissue infections frequently are associated with abscesses. Prompt surgical drainage of abscess is a key component of successful treatment.

Cellulitis *Purulent cellulitis, line-associated cellulitis, immunosuppression, or associated abscess should have Staph coverage. Rapidly progressive, pain out of proportion, shock – see necrotising fasciitis below*	Outpatient (Well-appearing, afebrile, no comorbidities or immunocompromise) *Nonpurulent (e.g. streptococcal)* Penicillin VK 500 mg PO Q6h (child: 25 mg/kg PO Q6h) Amoxicillin 500 mg PO TID (child: 15 mg/kg PO TID) *Purulent, possible MSSA* Cloxacillin 500 mg PO Q6h (child: 25 mg/kg PO Q6h) Cefalexin 500 mg PO Q6 (child: 10 mg/kg PO Q6h) *Purulent, possible MRSA* ADD to 'nonpurulent, strep' antibiotic Doxycycline 100 mg PO BID OR Co-trimoxazole 160/800 mg PO BID (child: 4 mg/kg/TMP PO BID) *Pen-allergic* Clindamycin 300–450 mg PO TID (child: 5–7 mg/kg PO TID)
	Inpatient Clindamycin 600 mg IV TID (child: 10 mg/kg IV Q6h) (Strep, MSSA, most MRSA) *Possible MSSA* Cloxacillin 2 g IV Q6h (child: 50 mg/kg IV Q6h) Cefazolin 1 g IV TID (child: 15 mg/kg IV TID) *Possible MRSA* Vancomycin 15–20 mg/kg IV BID (child: 10 mg/kg IV Q6h)
Diabetic foot infection *Multidisciplinary approach requiring antibiotics, wound care, surgical consultation, and limited to non-weight bearing. Consider underlying osteomyelitis*	*Mild:* see Outpatient cellulitis regimens above for dosing [Cefalexin OR cloxacillin OR clindamycin] AND [Co-trimoxazole OR doxycycline for MRSA coverage] *Moderate:* (MRSA, strep, gram-negative, anaerobic coverage) [Clindamycin 450 mg PO TID AND ciprofloxacin 500 mg PO BID] OR [Co-trimoxazole 160/800 mg PO BID AND amoxicillin-clavulanate 875 mg PO BID]

Necrotising fasciitis *Surgical emergency – requires aggressive debridement, cannot be treated with antibiotics alone*	Piperacillin/tazobactam 4.5 g IV TID OR ampicillin/sulbactam 3 g IV Q6h] AND Clindamycin 900 mg IV TID AND Ciprofloxacin 400 mg IV BID *Pen-allergic* [Ciprofloxacin 400 mg IV BID OR gentamicin 7 mg/kg IV QD] AND Metronidazole 500 mg IV TID AND Clindamycin 900 mg IV TID (if available) *Alternate* Benzylpenicillin 4 million units IV Q4h (child: 100,000 units/kg IV Q4h) AND Gentamicin 7 mg/kg IV QD AND Metronidazole 500 mg IV TID (child: 12.5 mg/kg IV TID) AND Clindamycin 900 mg IV TID (if available) child: 10 mg/kg IV Q6h) *Possible Staph aureus* Clindamycin as above. If clindamycin not available, ADD cloxacillin 2 gm IV 4h (MSSA) OR vancomycin 20 mg/kg IV BID (MRSA)
Deep space head and neck *Coverage includes oral flora, anaerobes, Strep species. Consider TB. If immunocompromised, consider Pseudomonas, Klebsiella. If post-operative or trauma, consider Staph, gram-negative rods*	Ampicillin-sulbactam 3 g IV Q6h (child: 100 mg/kg IV Q6h) OR [Ceftriaxone 2 g IV Q24h (child: 50 mg IV BID) AND metronidazole 500 mg IV TID (child: 12.5 mg/kg IV TID)] OR [Ciprofloxacin 400 mg IV BID + clindamycin 600 mg IV TID] *If possible MRSA* ADD vancomycin 15–20 mg/kg IV q12h (child: 10–15 mg/kg IV Q6h) *If possible Pseudomonas (immunocompromise)* SUBSTITUTE cefepime 2 g IV TID for ceftriaxone (child: 50 mg/kg IV TID)
Impetigo *Beta-haemolytic streptococci and Staphylococcus aureus* *If fever or systemic signs, consider Stevens-Johnson*	Localised, uncomplicated: mupiricin 2% ointment TID Bullous lesions: cloxacillin 500 mg PO Q6h (child: 25 mg/kg PO Q6h)
Dog or cat bite wound infection *Verify tetanus vaccination status. Assess need for rabies prophylaxis*	*Prophylaxis* Amoxicillin/clavulanic acid 875 mg PO BID or 500 mg PO TID (child: 8 mg/kg PO TID or 12.5 mg/kg PO BID) *Cellulitis* Metronidazole 500 mg PO TID (child: 12.5 mg/kg PO BID) AND Co-trimoxazole 160/800 mg PO BID (child: 4 mg/kg/TMP PO BID)

Urinary tract infections

UTI in women *Uncomplicated: well-appearing with normal VS, afebrile, no nausea/vomiting, minimal symptoms* *Complicated: recurrent infection, structural GU abnormalities, diabetes or immunocompromise, urinary stones* *Frequent antibiotic resistance in UTIs. Choose alternate antibiotic if treated within previous 3 months*	*Uncomplicated* Fosfomycine 3 g PO × 1 dose OR Co-trimoxazole 160/800 mg PO BID × 3 days OR Amoxicillin/clavulanic acid 875 mg PO BID × 5 days *Complicated* See Pyelonephritis regimens; treat × 7–10 days *Pregnancy and lactation* Amoxicillin/clavulanic acid 875 mg PO BID × 5 days OR Cefalexin 500 mg PO Q6h × 5 days OR Fosfomycine 3 g PO × 1 dose (asymptomatic bacteriuria in pregnancy or uncomplicated cystitis not in pregnancy only)
UTI in men *Consider prostatitis, urethritis in males with UTI. Test and treat for STIs if suspected*	*Outpatient* Co-trimoxazole 160/800 mg PO BID OR Ciprofloxacin 500 mg PO BID OR Fosfomycine 3 g PO Q3 days Treat × 10–14 days for uncomplicated UTI, 6 weeks for prostatitis.
Pyelonephritis and urosepsis *For pyelonephritis, consider 1x dose IV ceftriaxone, ciprofloxacin, or gentamicin prior to discharge on oral antibiotic* *Caution for gentamicin use in genitourinary infection unless current creatinine known and normal* *Change catheter in patients with indwelling Foley* *Consider local antibiotic resistance patterns, particularly when using fluoroquinolones*	*Pyelonephritis (fever, flank pain, no signs serious illness)* Ciprofloxacin 500 mg PO BID × 14 day OR Cefixime 200 mg PO BID × 14 days OR Cefalexin 500 mg PO Q6h × 14 days OR Co-trimoxazole 160/800 mg PO BID × 14 days *Urosepsis (ill-appearing, sepsis)* [Ceftriaxone 1 g IV QD ± gentamicin 7 mg/kg IV QD] OR [Ampicillin 2 g IV Q6h AND gentamicin 7 mg/kg IV QD] Transition to oral pyelonephritis regimen when clinically improved; min 3 days IV antibiotics, 14-day course total. *Possible Pseudomonas* (immunocompromise, indwelling Foley, history frequent UTIs or known GU abnormalities) Choose gentamicin or fluoroquinolone-based regimen as above *Possible Staph* (recent genitourinary surgical procedure) ADD Cloxacillin 500 mg IV Q6h (MSSA) OR vancomycin 15–20 mg/kg IV BID (MRSA)
UTI in children *Consider local antibiotic resistance patterns, particularly when using cefalexin, amoxicillin or ampicillin, co-trimoxazole* *Ampicillin-gentamicin regimen preferred for Enterococcus, Pseudomonas coverage in indwelling Foley, recent urinary tract instrumentation, known genitourinary abnormality* *Consider Staph infection if recent genitourinary surgical procedure*	*Uncomplicated* Preferred: cefpodoxime 10 mg/kg PO QD × 5–10 days OR Cefixime 16 mg/kg PO once, then 8 mg/kg PO QD × 5–10 days Alternate: cefalexin 10 mg/kg (max 500 mg) PO Q6h × 5–10 days OR Amoxicillin/clavulanic acid 8 mg/kg (max 500 mg) PO TID × 5–10 days OR Co-trimoxazole 4 mg/kg/TMP (max 160 mg) PO BID × 5 days *Pyelonephritis* [Ampicillin 50 mg/kg IV Q6h AND gentamicin 7.5 mg/kg IV QD] OR [Ceftriaxone 75 mg/kg IV QD ± gentamicin 7.5 mg/kg IV QD] ADD cloxacillin 50 mg/kg IV Q6h (MSSA) OR vancomycin 15–20 mg/kg IV Q6h (MRSA) if possible Staph

Syphilis

Note: penicillin is preferred agent. Data for alternate agents extremely limited; patients treated with alternate agents must be followed closely for rising titres/treatment failure.
Jarisch-Herxheimer reaction may occur in first 24 hours after penicillin with fever, shaking chills, low BP, worsening rash. Antipyretics, IVF and supportive care, close monitoring recommended.

Test all syphilis patients for HIV. Test and treat sexual partners for syphilis; consider other STIs. *(p. 356)*
Management of syphilis frequently is complicated and requires prolonged patient follow-up. Strongly consider specialist consultation for assistance.

Primary, secondary, early latent syphilis *Only if able to verify syphilis infection within the previous 1 year*	Preferred: benzathine penicillin 2.4 million units IM × 1 dose Pen-allergic: doxycycline 100 mg BID
Late latent syphilis or latent (asymptomatic) syphilis of unknown duration	Benzathine penicillin 2.4 million units IM weekly × 3 doses Penicillin sensitivity, not type 1 allergic reaction: consider ceftriaxone 1 g IV QD × 14 days. Preferred is admission with penicillin desensitisation, when available. Gummatous syphilis may use doxycycline 100 mg PO BID × 28 days
Syphilis in pregnancy	Screen all pregnant women at first visit, then repeat screening in third trimester and at delivery. Classify and treat as above.

Congenital syphilis *Evaluate severity of disease: CXR, long bone XR, liver function, ophthalmologic exam, hearing evaluation, CSF (VDRL, WBC, protein), CBC, and neuroimaging* *Obtain baseline RPR or VDLR titer and CSF prior to initiating treatment* *If > 1 day treatment missed, start over full course*	*Presentations and patient factors vary considerably. Expert recommendations allowing for out-patient treatment in certain cases exist, but depend upon reliable patient follow-up and serial testing. Out of concern for poor follow-up or limited testing, we have offered the most conservative treatment strategies* Treat any neonate with a suggestive exam, titer greater than the mother's, positive umbilical cord/placenta/body fluid treponemal testing, maternal history of inadequate treatment (including treatment with non-penicillin agent) or treatment within 4 weeks of delivery < 1 month age: < 8 days age: benzylpenicillin 50 000 units/kg IV BID × 10 days 8 days to 1 month: benzylpenicillin 50 000 units/kg IV TID × 10 days > 1 month age: Benzylpenicillin 50 000 units/kg IV Q4h × 10 days, then benzathine penicillin 50 000 units/kg IM × 1 on day 11 Monitoring: Titer Q3 months × 6 months. If titer not falling at 3 mos or not negative at 6 months, treatment failure. Test CSF and repeat treatment as above If neurosyphilis, repeat CSF WBC at 6 months. If CSF WBC elevated, repeat treatment
	At risk: normal exam, titer less than maternal titer, mother received adequate treatment > 4 weeks prior to delivery or prior to pregnancy with no evidence relapse. Penicillin 50 000 units/kg IM × 1
Treatment failure *Follow RPR or VDRL for titer. Do not follow fluorescent treponemal antibody tests*	Primary, secondary syphilis: monitor treponemal antibody titres at 6 and 12 months Latent syphilis: antibody titer every 6 months × 2 years Treatment failure: failure of titer to fall by at least four-fold (two dilutions) from baseline. Resume treatment and administer late latent syphilis regimen, consider neurosyphilis testing Neurosyphilis and HIV-associated, see below
Neurosyphilis or cardiovascular syphilis	Benzylpenicillin 3–4 million units IV Q4h × 14 days (minimum) ± Benzathine penicillin 2.4 million units IM day 15, repeat weekly × 3 IM doses (optimal) Mild pen-allergy: ceftriaxone 2 g IV QD × 14 days Severe pen-allergy: admission, penicillin desensitisation Monitoring: CSF WBC Q6 months. If WBC should be falling at 6 months and normal at 2 years. If not, treatment failure. Monitor serum titres as above
Syphilis in HIV	Treatment recommendations as above. Literature suggests slower fall in titer in HIV+. ARVs may improve therapeutic response. Obtain baseline titer day 1 of treatment. Monitoring: Primary, secondary syphilis: titer Q3 months × 1 year Early or late latent syphilis: titer Q6 months × 2 years Neurosyphilis: CSF testing (WBC) Q6 months; if still positive at 2 years, repeat neurosyphilis treatment. Treatment failure: If titer has not fallen by at least four-fold by end of monitoring period, if titer rises four-fold or more during monitoring, or patient develops symptoms, treatment failure. Perform LP with CSF evaluation for syphilis. If CSF (+), treat as neurosyphilis. If CSF (-), treat as late latent syphilis. Continue monitoring titer as above.
Diarrhoea *Mainstay of therapy is supportive care: nutrition, hydration, electrolytes. Treat extra-intestinal infections. Avoid anti-motility agents; prolong shedding, may worsen disease.* *Preference for PO antibiotics in enteric infections, unless severe illness limits absorption or ability to take oral.*	
Acute diarrhoea in adults	*Acute watery:* supportive care, consider cholera *Acute bloody (dysentery):* majority dysentery caused by *Shigella* 1. Treat shigellosis based on local sensitivities. IV therapy preferred for immunodeficiency, inability to take PO, severe disease (temperature > 39 or < 35, elevated WBC, lethargy, shock) Preferred: ciprofloxacin 400 mg IV or 500 mg PO BID Alternate IV: ceftriaxone 1 g IV QD Alternate PO: azithomycin 500 mg PO QD 3-day course, extend to 7 days if severely ill or immunosuppressed 2. If no improvement at 2 days, switch to alternate Sheila agent 3. ADD campylobacter treatment if high fever, immunosuppressed, severe disease, dysentery > 1 week Azithomycin 500 mg PO/IV QD OR Ciprofloxacin 400 mg IV or 500mg PO BID; beware cipro resistance 3-day course, extend to 7 days if severely ill or immunosuppressed 4. ADD empiric anti-parasitic (entamoeba) if persistent dysentery after 2 antibiotics Metronidazole 500–750 mg PO/IV TID 7–10 day course Broad-spectrum antibiotics for abdominal sepsis if peritonitis

<table>
<tr><td>Acute diarrhoea in children</td><td>Acute watery: consider mild cholera if known outbreak
Acute bloody (dysentery): majority dysentery caused by Shigella
1. Treat shigellosis based on local sensitivities. IV therapy preferred for immmunodeficiency, inability to take PO, severe disease (temperature > 39 or < 35, elevated WBC, lethargy, shock)
Ceftriaxone 50 mg/kg IV QD
Azithomycin 10 mg/kg PO QD
Alternate: ciprofloxacin 10–15 mg/kg IV (only if other agents unavailable/resistance)
3-day course, extend to 7 days if severely ill or immunosuppressed
2. No improvement at 2 days, switch to alternate Shigella agent
3. ADD treatment for Campylobacter if high fever, immunosuppressed, severe disease, dysentery > 1 week
Azithomycin 10 mg/kg PO/IV QD (child)
Alternate: ciprofloxacin 15 mg/kg PO BID only if other agents unavailable; beware cipro resistance
3-day course, extend to 7 days if severely ill or immunosuppressed
4. ADD empiric anti-parasitic (entamoeba) if persistent dysentery after 2 antibiotics
Metronidazole 12–18 mg/kg PO/IV TID (35–50 mg/kg/day divided)
7–10 day course
Broad-spectrum antibiotics for abdominal sepsis if peritonitis
A minority of dysentery cases is caused by enterohaemorrhagic E. coli 0157:H7. Controversy exists as to antibiotics worsening toxin-related renal failure. Monitor closely</td></tr>
<tr><td>Chronic diarrhoea</td><td>Unexplained persistent diarrhoea > 1 month meets WHO criteria for HIV clinical stage 3</td></tr>
<tr><td>HIV
All HIV patients with diarrhoea merit full investigation
Immunosuppression may require prolonged course; subject to relapses. Mainstay of treatment is restoration of immune function with ARVs
Beware diarrhoeal co-trimoxazole resistance in HIV+ on co-trimoxazole prophylaxis</td><td>Acute
Acute watery diarrhoea: supportive care
Acute bloody diarrhoea: see above adult and paediatric recommendations
Chronic diarrhoea: effective ARV therapy mainstay of treatment
Entaemoba
Metronidazole 500–750 mg TID (adult); 12–18 mg/kg TID (total 35–50 mg/kg/d) (child); PO strongly preferred; 10-day course minimum
After metronidazole, give luminal agent: paromomycin 10 mg/kg TID PO × 7 days OR diloxanide furoate 500 mg (child: 6.5 mg/kg) PO TID × 10 days
Giardia: metronidazole 500–750 mg (child: 12–18 mg/kg) TID; PO strongly preferred; 10-day course minimum
Cryptosporidium: symptomatic management
Cyclospora: co-trimoxazole 160 mg/800 mg (child: 4 mg/kg/TMP) PO BID × 10 days
Isospora: limited treatment evidence
Adults: co-trimoxazole 160/800 mg PO BID AND ciprofloxacin 500 mg PO BID × 10 days. Alternate: co-trimoxazole 160/800 mg PO Q6h × 10 days, then 160/800 mg PO BID × 3 weeks
Children: co-trimoxazole 4 mg/kg/TMP PO Q6h × 10 days, then 4 mg/kg/TMP BID × 3 weeks. Consider ciprofloxacin addition as above adult regimen if severe or refractory disease
Microsporidium: possible efficacy with albendazole 800 mg PO QD (child: 7.5 mg/kg PO BID)</td></tr>
<tr><td colspan="2">Tuberculosis
Stages and types of disease, patterns of resistance vary widely. Refer to national and local guidelines for diagnosis, treatment, reporting. Use directly-observed therapy programmes. Acute TB care may involve initial diagnosis and management of complications (i.e. pleural effusion, ascites, tamponade, PTX, cord compression, pathologic fracture). Initiation of anti-TB medications may be urgent but is rarely emergent. Do so only with specialist consultation and/or standardised guidelines, admission or ensured follow-up in order to ensure DOTS.
More detailed regimens may be found with:
World Health Organization. 2009. Treatment of tuberculosis guidelines. 4rd edition. Geneva: WHO.
World Health Organization. 2004. TB/HIV: A clinical manual. 2nd edition. Geneva: WHO.</td></tr>
<tr><td>CNS/Meningitis</td><td>See Meningitis section above</td></tr>
</table>

Notes on antibiotic selection

- **Amoxicillin/clavulanic acid:** multiple preparations exist. Dosing given in milligrams of amoxicillin component. Frequency depends upon preparation/dosage prescribed (e.g. 500 mg PO TID, 875 mg PO BID).
- **Cephalosporins and penicillin allergy:** many patients with penicillin allergy are able to tolerate late-generation cephalosporins (**ceftriaxone, cefepime, cefpodoxime**), and their use may be reasonable if not viable alternate agent exists. If used in these patients, administer a partial dose, monitor 1 hour, then administer remainder dose if no reaction. Patients with type 1 allergic response (true hives, anaphylaxis) should *not* receive cephalosporins.
- **Chloramphenicol:** refer to regional resistance patterns. Risk for aplastic anaemia, thrombocytopenia, leukopenia. Avoid in children under 2 weeks due to ‘grey baby’ syndrome. Avoid in last one-month of pregnancy and first month of breastfeeding.
- **Fluoroquinolones (ciprofloxacin, levofloxacin, moxifloxacin):** contraindicated in pregnancy, breast-feeding

and in children due to possible tendon and cartilage damage in weight-bearing joints. No conclusive data exists. Use alternate agent when possible. Consider risk/benefit when alternate not available or fluoroquinolone first-line, especially in severe disease.

- **Gentamicin:** avoid if known renal insufficiency. Caution in use for genitourinary infections; use only if creatinine known to be normal.
- **Sulfa (co-trimoxazole, TMP/SMX) and nitrofurantoin:** caution for haemolysis in G6PD-deficiency.
- **Sulfa (co-trimoxazole, TMP/SMX):** caution in pregnancy due to increased risk of congenital defects (first trimester) and risk for neonatal kernicterus (last month of pregnancy, first month breastfeeding). Use alternate agent when possible.
- **Tertracyclines (doxycycline):** contraindicated in pregnancy, breastfeeding and in children due to dental staining. Effect is dose-related, rarely occurs with single course of treatment, and primarily in those under age 8. Mild cosmetic risk may outweigh benefit in tick fever or settings in which other antibiotic agents unavailable.
- **Vancomycin, cloxacillin, and staphylococcus**: vancomycin, clindamycin, and cloxacillin have been recommended as anti-staphylococcal agents. In general, staphylococcus in much of Africa remains cloxacillin susceptible (MSSA). However, providers should be aware of the global rise of methicillin-resistant staph aureus (MRSA) and treat to local resistance patterns. Vancomycin should be reserved for severe infections; mild to moderate infections can be treated with clindamycin, doxycycline, or co-trimoxazole.
- **Penicillin-resistant Streptococcus:** increasing penicillin resistance requires higher doses of penicillins and cephalosporins, and/or use of alternative agents, such as vancomycin. As with MRSA, these strains currently are believed to be relatively rare in Africa. However, providers should be aware and follow emerging resistance patterns.

Dermatologic therapies

Skin conditions may be treated with topical and/or systemic therapies. The effectiveness of topical medications is dependent on the drug, the vehicle of transport, the concentration of medication and the anatomic location being treated. Avoidance of triggering factors, such as allergens or irritants, is essential.

Topical vehicles

The vehicle is the substance into which the active ingredient is mixed. Any active ingredient can be mixed into any vehicle. The following are commonly used vehicles:

Ointments – lubricating and greasy
  Example: white petroleum
  Use: smooth hairless skin, dry lesions, thick skin areas
Creams – not as greasy and disappear when massaged into skin
  Example: hydrocortisone 1% cream
  Use: topical inflammation
Lotions – pourable and have little residue
  Example: triamcinolone 0.1% lotion
  Use: hairy areas
Oils – less irritation than other preparations
  Example: fluocinolone 0.01% oil
  Use: scalp, especially in coarse or curly hair
Jelly – greaseless and quick drying
  Example: lidocaine 2% jelly
  Use: topical anaesthesia

Topical medication

Corticosteroids

Topical corticosteroids are useful for conditions which involve immune response and inflammation; they may provide symptomatic relief for burning lesions (e.g. haemorrhoids).

Systemic side effects are rare but may occur with prolonged use of high potency topical corticosteroids. Side effects are more common with long term use of high potency corticosteroids (these changes may be irreversible) and include skin atrophy / telangiectasias / striae, acne and hypopigmentation.

Drug selection:

- Very high potency: severe skin conditions on non-facial and non-opposing surfaces (i.e. soles, palms)
- Medium to high potency: mild to moderate skin conditions on non-facial and non-opposing surfaces
- Low potency: for large surface area and thin skin (face, eyelids, genitals, and opposing surfaces).

Avoid high and very high potency preparation on the face, in the flexures and in the treatment of rashes on babies.

Concentration	Corticosteroid	Dosage Form	Comments
0.05 %	Betamethasone	Cream, gel, lotion, ointment	Very high potency
0.05 %	Clobetasol	Cream, gel, lotion, ointment	Limit treatment to less than 3 weeks
0.05%	Fluocinonide	Cream, gel, ointment	High potency
0.05%	Desoximetasone	Cream, ointment	Limit treatment to less than 6–8 weeks
0.1%	Triamcinolone	Cream, lotion, ointment	Medium potency
0.1%	Mometasone	Cream, lotion, ointment	Limit treatment to less than 6–8 weeks
1 %	Hydrocortisone	Cream, ointment, solution	Low potency
0.05%	Desonide	Cream, ointment	Limit treatment to resolution of symptoms
0.01%	Fluocinolone	Cream, solution	

Antifungals

Topical antifungals are generally safe, effective, and have limited systemic absorption. They are the preferred treatment for uncomplicated superficial fungal infections. In general, hair and nail fungal infections do not respond to topical antifungal treatment and must be treated systemically. These drugs often have to be used for prolonged courses and completion of treatment is essential.

Examples of topical antifungal agents:
Clotrimazole, econazole, ketoconazole, miconazole, nystatin, terbinafine.

Topical and systemic medications used for treatment of *Acne vulgaris*

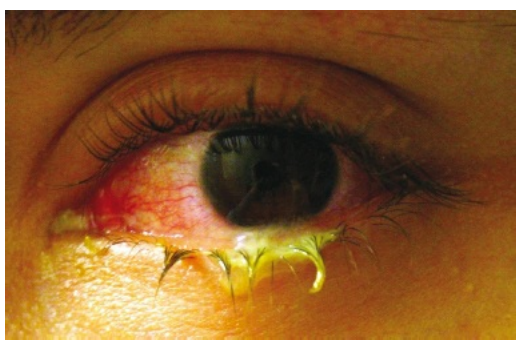
- Benzoyl peroxide
  - » An antibacterial and comedolytic topical agent
  - » A common side effect of the medication is inadvertent bleaching of hair or clothing, skin irritation, and skin peeling
- Topical antibiotics
  - » Usually used in combination with benzoyl peroxide
  - » Examples include erythromycin gel, clindamycin gel, and tetracycline ointment
- Topical tretinoin and systemic isotretinoin
  - » Vitamin A derivatives which inhibit keratinocytes and microcomedone formation
  - » Systemic isotretinoin is indicated for severe acne failing other therapies
  - » Causes birth defects and is absolutely contraindicated in female patients who may be pregnant
  - » Female patients of childbearing age should use two forms of contraception during treatment
  - » Side effects include dry lips and skin, liver dysfunction, and elevated triglycerides.

Images

Oral hairy leukoplakia

Oral thrush

Koplik's spots

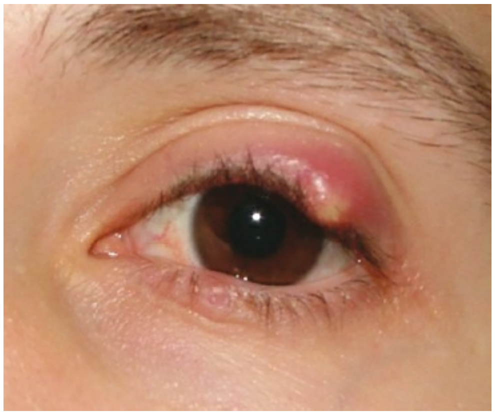


Bacterial conjunctivitus

Subconjunctival haemorrhage

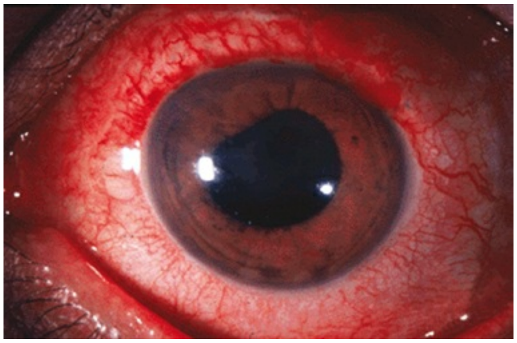
Corneal abrasion

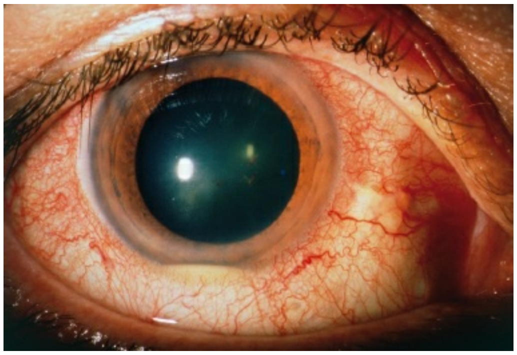
Stye

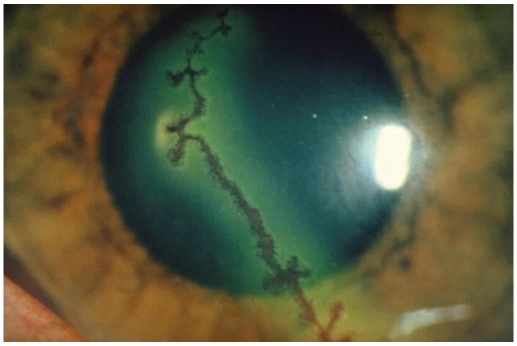


Chalazion


Episcleritis

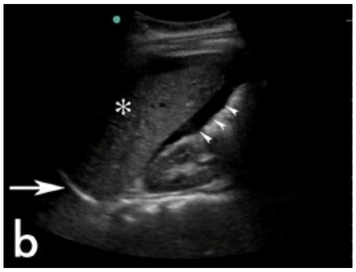

Anterior uveitis


Hypopyon


Dendritic ulcer

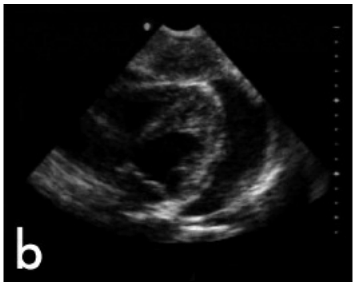
FAST Negative
Morison’s pouch view including the diaphragm (arrow), liver, and kidney

Source: Used with permission from the Partners in Health Manual of Ultrasound for Resource Limited Settings



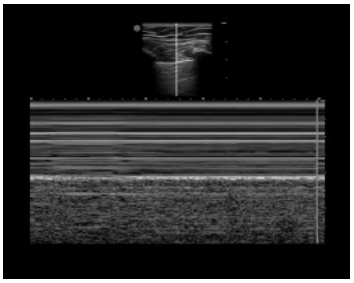
FAST Positive
Note blood in Morison’s pouch (arrowheads)

Source: Used with permission from the Partners in Health Manual of Ultrasound for Resource Limited Settings



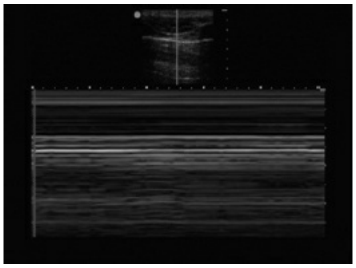
Pericardial effusion subxiphoid

Source: Used with permission from the Partners in Health Manual of Ultrasound for Resource Limited Settings



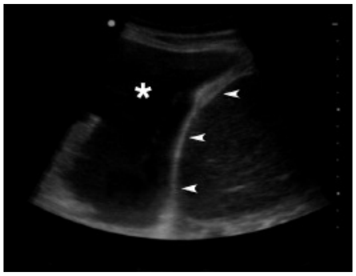
M mode tracing of normal lung (‘waves on a beach’)

Source: Used with permission from the Partners in Health Manual of Ultrasound for Resource Limited Settings



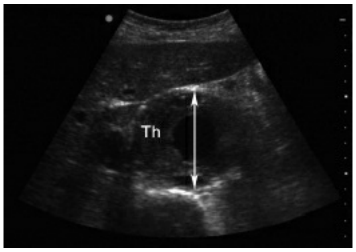
M mode tracing of a pneumothorax

Source: Used with permission from the Partners in Health Manual of Ultrasound for Resource Limited Settings



Pleural effusion (*) overlying the diaphragm (arrowheads)

Source: Used with permission from the Partners in Health Manual of Ultrasound for Resource Limited Settings



AAA with thrombus (Th) in vessel wall

Source: Used with permission from the Partners in Health Manual of Ultrasound for Resource Limited Settings

Probe position for RUQ view with the index marker pointing toward the patient's head (arrow)

Source: Used with permission from the Partners in Health Manual of Ultrasound for Resource Limited Settings

Probe position for LUQ view

Source: Used with permission from the Partners in Health Manual of Ultrasound for Resource Limited Settings

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Page numbers in *italics* point to illustrations, including rapid assessment protocols

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